A STUDY ON MATERNAL MORBIDITY AND MORTALITY IN JAUNDICE COMPLICATING PREGNANCY

TAMILNADU

DR. MGR MEDICAL UNIVERSITY,

CHENNAI

Dissertation Submitted to in partial fulfillment of the requirements for the degree of

M. S. (OBSTETRICS AND GYNAECOLOGY)

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

COIMBATORE MEDICAL COLLEGE HOSPITAL,

COIMBATORE

APRIL 2015

DECLARATION

I hereby declare that this dissertation entitled **"A STUDY ON MATERNAL MORBIDITY AND MORTALITY IN JAUNDICE COMPLICATING PREGNANCY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr S. BAMA M.D .D.G.O.,** Associate Professor, Department of Obstetrics & Gynaecology, Coimbatore Medical College & Hospital, Coimbatore.

Date :

Place :

Dr.T.GUHAPREETHA

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This is to certify that the dissertation entitled **A STUDY ON MATERNAL MORBIDITY AND MORTALITY IN JAUNDICE COMPLICATING PREGNANCY"** is a bonafide and genuine research work Carried out by **Dr. T. GUHA PREETHA** in partial fulfilment of the requirement for the degree of Master of Surgery in Department of Obstetrics & Gynaecology.

Date :

Guide
Dr.S.Bama M.D.D.G.O, Associate Professor
Department of Obstetrics & Gynaecology
Coimbatore Medical College Hospital.

Date :

Dr. P. Sundari M.D., D.G.O
HOD,
Department of Obstetrics & Gynaecology
Coimbatore Medical College Hospital.

Date :

Dr. Revwathy, M.D., D.G.O., DNB
The Dean,
Coimbatore Medical College Hospital.

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : T. GUHA PREETHA

Course : M.S [O&G]

Period of Study : 2012 - 2015

College : COIMBATORE MEDICAL COLLEGE

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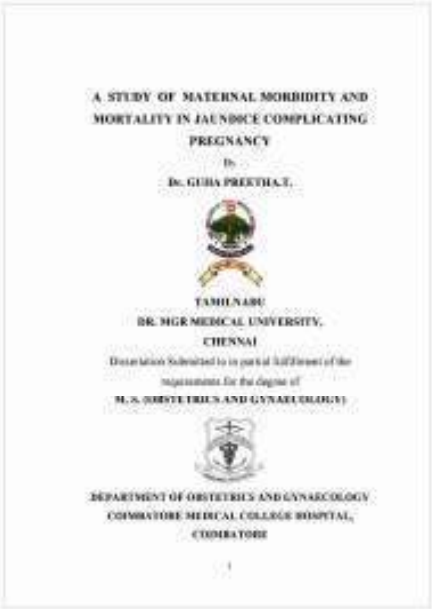

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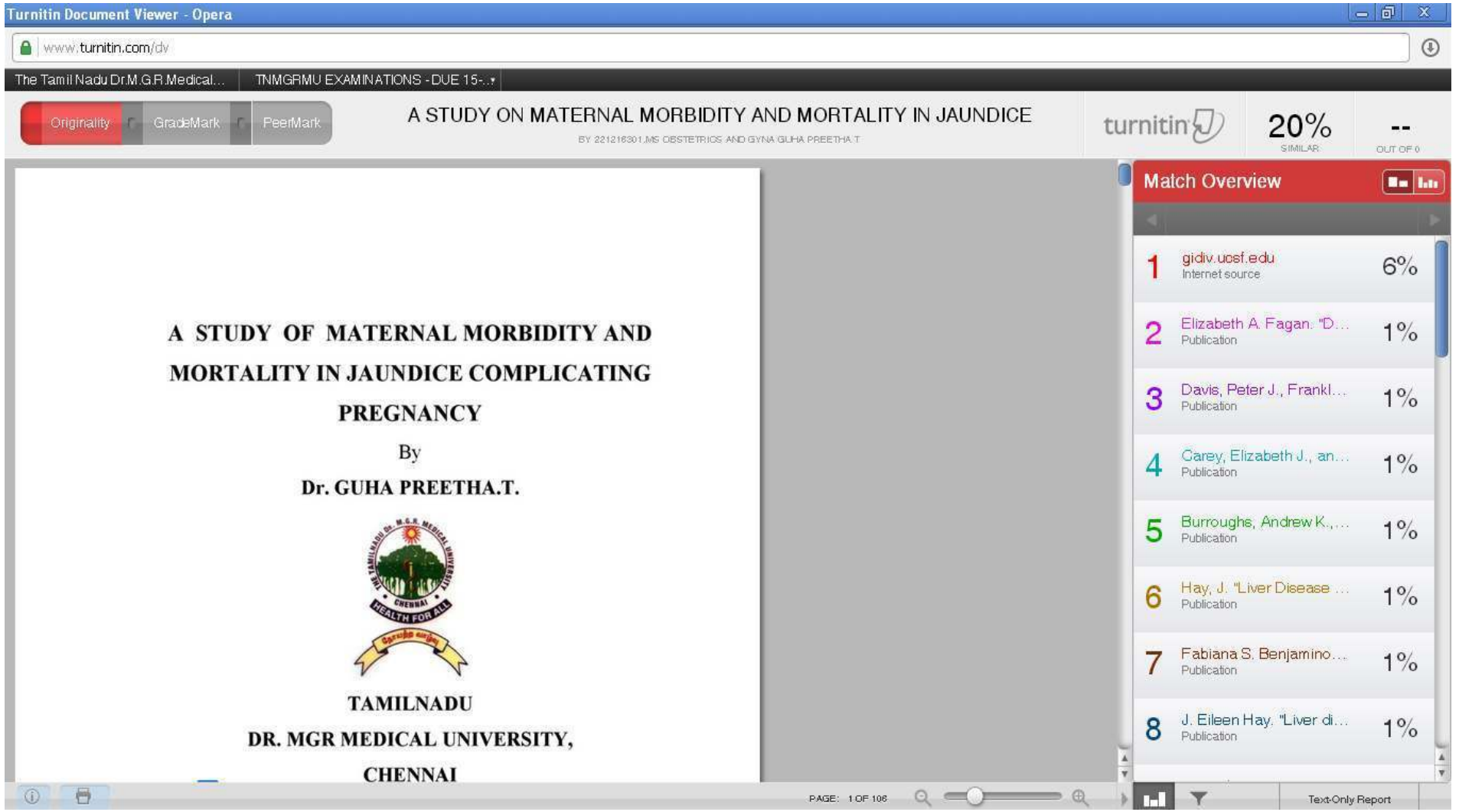
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Date: **Signature of the Candidate**

Place: Coimbatore **Name: Dr. GUHA PREETHA T**

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ABBREVIATIONS

AFLP	–	Acute Fatty Liver of Pregnancy
ALT	–	Alanine Transaminase
AST	–	Aspartate Transaminase
DIC	–	Disseminated intravascular coagulation
FAO	–	Fatty acid oxidation
G	–	Gravida
GGT	–	Gamma Glutamyl Transferase
HAV	–	Hepatitis A virus.
HBIG	–	Hepatitis B Immunoglobulin
HBsAG	–	Hepatitis B surface antigen.
HBV	–	Hepatitis B Virus
HCV	–	Hepatitis C virus
HELLP	–	Hemolysis, Elevated liver enzymes, Low platelet.
HEV	–	Hepatitis E virus
HIV	–	Human immunodeficiency virus.
HSV II	–	Herpes simplex virus II
HUS	–	Hemolytic uremic syndrome
IHCP	–	Intra hepatic cholestasis of pregnancy
IUGR	–	Intra uterine growth restriction.
LCHAD	–	Long chain 3-hydroxy acyl –COA dehydrogenase
LDH	–	Lactate dehydrogenase
P	–	Parity
RBC	–	Red blood cell
TTP	–	Thrombotic thrombocytopenic purpura
UDCA	–	Urso deoxy cholic acid
WBC	–	White blood cell

INTRODUCTION

Jaundice in pregnancy carries a grave prognosis and is responsible for 10% of maternal deaths. Abnormal liver function test occur in 3% to 5% of Pregnancy.

The causes may be

1) Co-incidental –viral Hepatitis, Gall stones.

2) Underlying chronic liver disease – cirrhosis, portal hypertension,chronic viral hepatitis, cholestatic jaundice.

3) Pregnancy related

   i) Associated with Pre eclampsia:-

      Preeclampsia

      HELLP syndrome

      Acute fatty liver of pregnancy

   ii) Without pre eclampsia :

      Hyperemesis gravidarum

      Intrahepatic cholestasis of pregnancy

4) Physiological changes during pregnancy –

Abnormal liver function test may occur as physiological change in pregnancy.

In developing countries like India, there is high mortality and morbidity in jaundice complicating pregnancy is due to associated factors like lack of awareness, poor hygiene and sanitation,malnutrition, anaemia, unbooked cases, late referral in moribund condition.

The common cause of death is due to Hepatic encephalopathy, shock,disseminated intravascular coagulation, renal failure, postpartum haemorrhage.

The aim of this study is to analyse the various causes for jaundice in pregnancy, the factors affecting its course and to determine the outcome of pregnancy among the pregnant women treated at Government Coimbatore Medical College and Hospital, Coimbatore.

AIMS AND OBJECTIVES

- To study the incidence and distribution of jaundice in relation to age, parity, socio economic status and duration of pregnancy.
- To study the relation of serum bilirubin levels to maternal mortality.
- To assess the severity of maternal outcome in terms of maternal mortality and morbidity.

ANATOMY OF LIVER

Liver is the largest exocrine gland in the body weighing about 1.5 kg in an average adult weighing about 70 kg. This is located in the right hypochondrium and a part of epigastric region.

Liver is attached to the anterior abdominal wall and the diaphragm by four distinctive ligaments:

1. Coronary ligament which connects the posterior surface of the right hepatic lobe to the diaphragm with a superior and an inferior layer between that lies in the bare area of the liver;

2. Right triangular ligament which is formed by fusion of the superior and inferior layers of the coronary ligament;

3. Left triangular ligament which connects the posterior surface of the left lobe of the liver to the diaphragm

4. Falciform ligament which extends from the diaphragm and anterior wall above the level of the umbilicus to the surface of the liver, where it divides the left hepatic lobe into the left lateral and left medial segments

Liver segment is divided into eight segments . COUINAUD coined a system for liver segmental nomenclature (8 segments). Liver is divided into segments by a longitudinal planes drawn through each hepatic vein to the vena cava and a transverse plane at the level of the main portal bifurcation .Cantlie's line marks the course of the middle hepatic vein

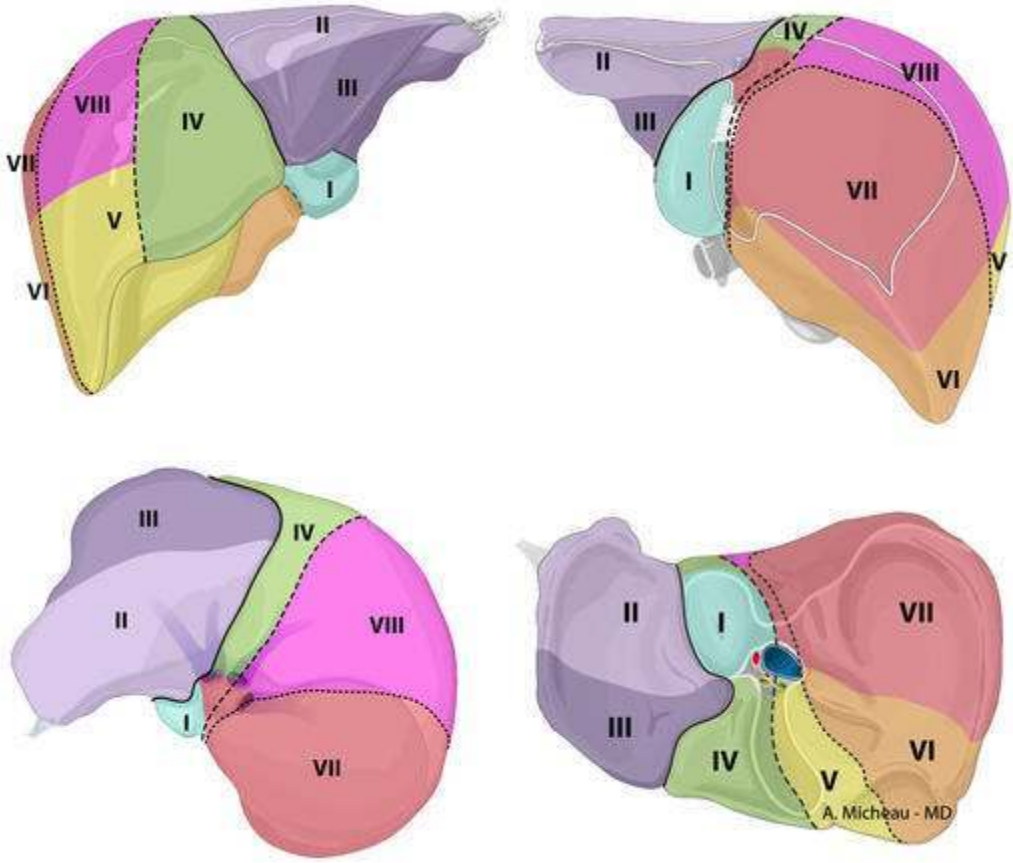


Fig. 1. Segments of Liver

BLOOD SUPPLY

The two sources that supply blood to the liver are Hepatic artery and portal vein. Hepatic artery is a branch from celiac trunk of aorta . Portal vein is formed by the confluence of the superior mesenteric vein and the splenic vein at the level of the second lumbar vertebra behind the head of pancreas . It supplies about 75% of the total liver blood supply by volume.

Blood exits the liver via the central vein accounting for 25% of cardiac output. Blood flow into the liver is controlled by number of factors like Muscular sphincters , autonomic nervous system, circulating hormones, bile salts, and metabolites .

VENOUS DRAINAGE

Majority of the venous drainage of the liver occurs through three hepatic veins . Right hepatic vein drains the segments 6,7,8 and enters directly into the vena cava.

Middle hepatic vein drains segments 5 and inferior part of segment 4 . Left hepatic vein drains the segments 2,3 and superior part of 4.

MICROANATOMY

Liver is composed of hexagonal shaped units called hepatic lobules .Each lobule is a tiny hexagonal or pentagonal cylinder of about 2x1 mm. These form the anatomical units of the liver. A tributary of hepatic vein extends through the centre of each lobule called interlobular vein (central vein).

Around this central vein hepatic cells are arranged as plates or irregular walls radiating outward. On the outer corners of each lobule , portal triad consisting of a branch of portal vein , a branch of hepatic artery and an interlobular bile ductules is arranged. Three adjoining parts of hepatic lobules constitute portal lobule with common drainage of bile into bile ductules of portal triad.

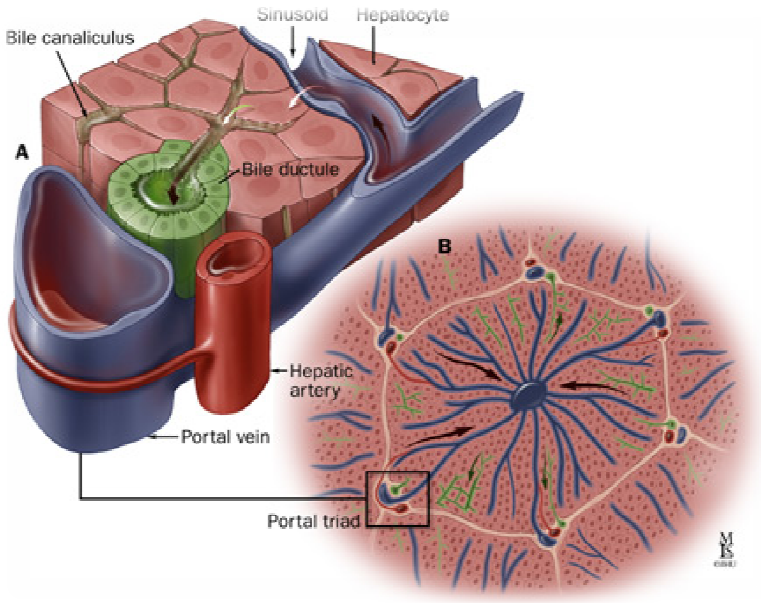


Fig. 2. Portal Triad

PORTAL TRIAD

Branches of portal vein, hepatic artery and the biliary ducts bound together in the perivascular fibrous capsule to form portal triad. Hepatocytes are arranged in plates joined with tight junctions and the apical membrane forms the biliary canaliculi. Hepatocytes are segregated from the blood-filled sinusoids by fenestrated endothelial cells without a basement membrane, and by a loose connective tissue layer known as the space of Disse.

HEPATIC STELLATE CELLS

These are Star shaped cells that reside in the space of Disse. It helps to Store lipids particularly vitamin A.

Inflammatory cytokines cause activation, which involves the loss of stores of vitamin A and a dramatic upregulation in the production of extracellular matrix materials, such as collagen. When collagen is deposited in the space of Disse it impairs hepatic function.

KUPFFER CELLS

Kupffer cells belongs to macrophage lineage. The sinusoids are lined by kupffer cells which clear the cellular debris and other particulate material and to some extent bacteria that enters the portal triad.

They express cell-surface receptors for altered proteins. Fc immunoglobulin receptors used to internalize foreign proteins or micro-organisms that have been coated with host antibodies.

BILIARY SYSTEM

Gallbladder is a biliary reservoir that lies against the inferior surface of segments IV and V of the liver, usually making an impression against it. A peritoneal layer covers most of the gallbladder except for the portion adherent to the liver. The size is variable, but usually about 10 cm long and 3 to 5 cm wide. Gallbladder is composed of a fundus, body, infundibulum, and neck . Ultimately empties into the cystic duct

BIOCHEMICAL FUNCTIONS OF LIVER

1. Storage fuction- stores protein , vitamins, glycogen and folic acid.
2. Synthesis of plasma proteins , glycogen , phospholipids , bile acids and heparin .
3. Secretory function-secretes bile acids and bile pigments into the bile.
4. Metabolic function-metabolism of protein, carbhohydrate and fat.

5. Excretory function-excretion of hormones, cholesterol, heavy metals and bile pigments.
6. Drugs detoxification.
7. Production of WBC and RBC during fetal life.
8. Defence mechanism carried out by kupffer cells.

PRODUCTION AND METABOLISM OF BILIRUBIN:

SOURCE OF BILIRUBIN:

Mainly 80% is from senescent RBC and about 15 -20 % from ineffective erythropoiesis .

Metabolism of haem containing protein can be divide into three phases:

(i) Hepatic uptake

(ii) Conjugation

(iii) Excretion into bile (rate limiting step)

UPTAKE:

Albumin bound unconjugated bilirubin enters liver and the complex dissociates. Non-polar bilirubin enters the hepatocyte by diffusion . It binds to cytoplasmic anion binding protein ligandin

glutathione-s-transferase and prevents efflux of bilirubin back into plasma.

CONJUGATION:

Unconjugated bilirubin gets conjugated with glucuronic acid forming water soluble bilirubin glucoranide ,catalysed by glucuronyl transferase.

Conjugation occurs in endoplasmic reticulam.

EXCRETION:

Water soluble conjugated bilirubin is excreted into the biliary canaliculi by the hepatocytes . In gut it is converted to stercobilinogen by bacteria and Oxidized to stercobilin which is colored. Excreted in feces. Some stercobilin may be re-adsorbed by the gut and re-excreted by either the liver or kidney

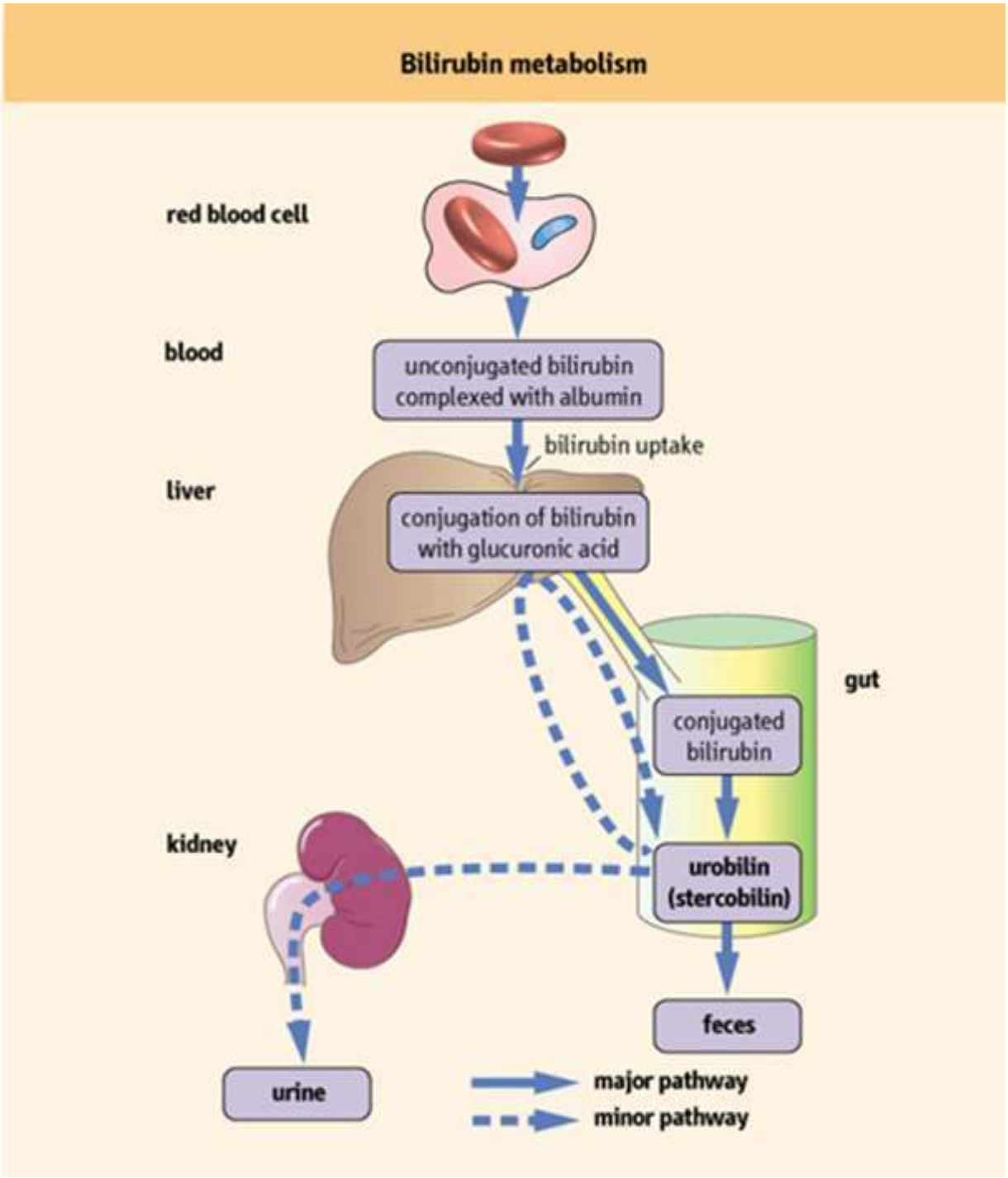


Fig. 3. Bilirubin Metabolism

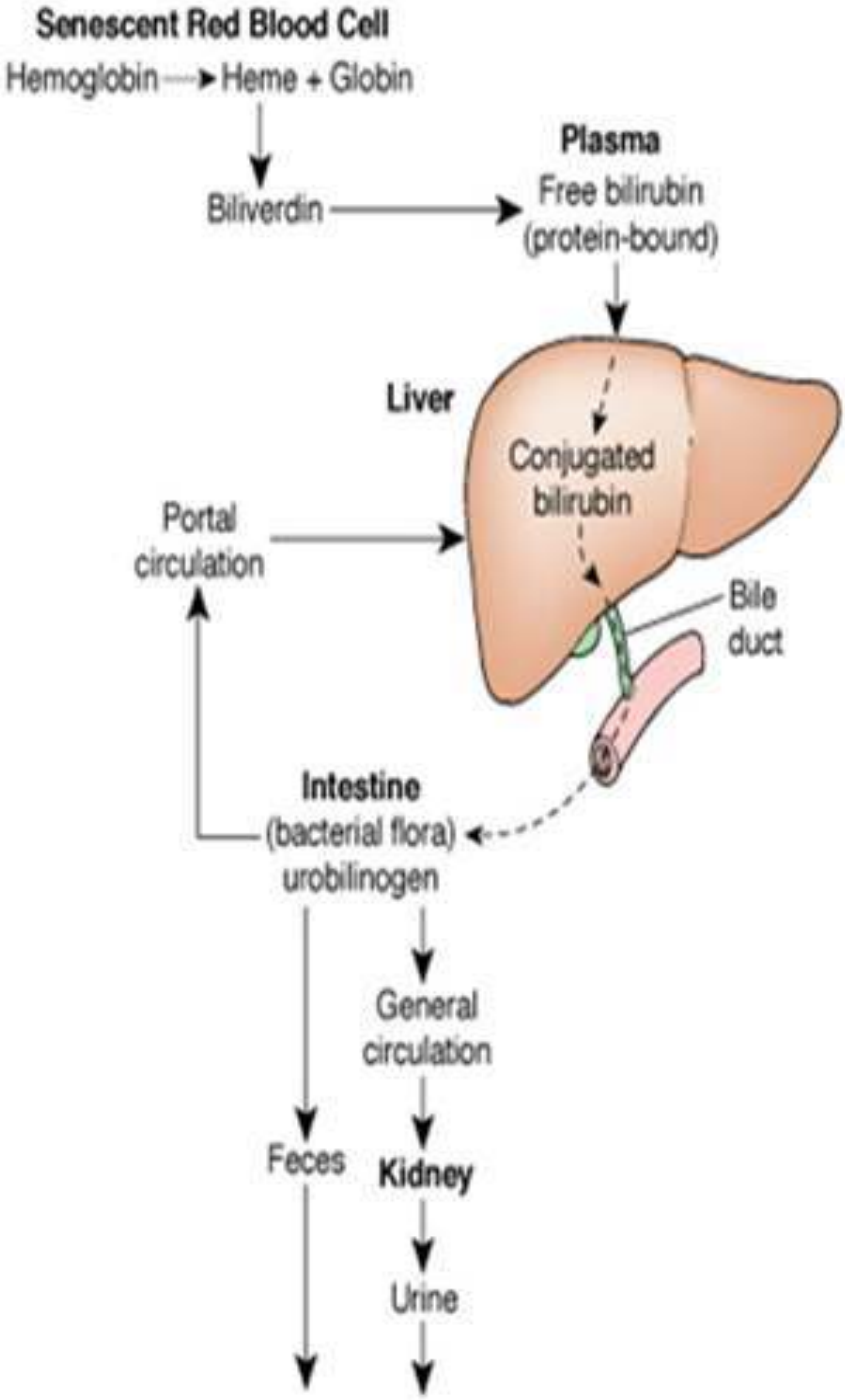


Fig. 4. Bilirubin Metabolism

Causes of Unconjugated hyperbilirubinaemia:

1. Increased bilirubin formation e.g., Haemolysis , Ineffective erythropoiesis , Blood transfusion and Haematoma.

2. Decreased bilirubin uptake by hepatocyte e.g.,Drugs like Rifampicin, Gilbert's syndrome

3. Deficit in conjugation -Gilbert's syndrome , Crigler Najjar syndrome , Drugs

Diseases of Unconjugated hyperbilirubinaemia:

1) Dubin-Johnson syndrome, Rotor's syndrome
2) Hepatocellular dysfunction
3) Hepatic disorder with prominent cholestasis
4) Biliary duct obstruction.

PREHEPATIC JAUNDICE

Prehepatic jaundice results from excessive RBC lysis as in haemolytic anaemia . Unconjugated bilirubin level is increased.

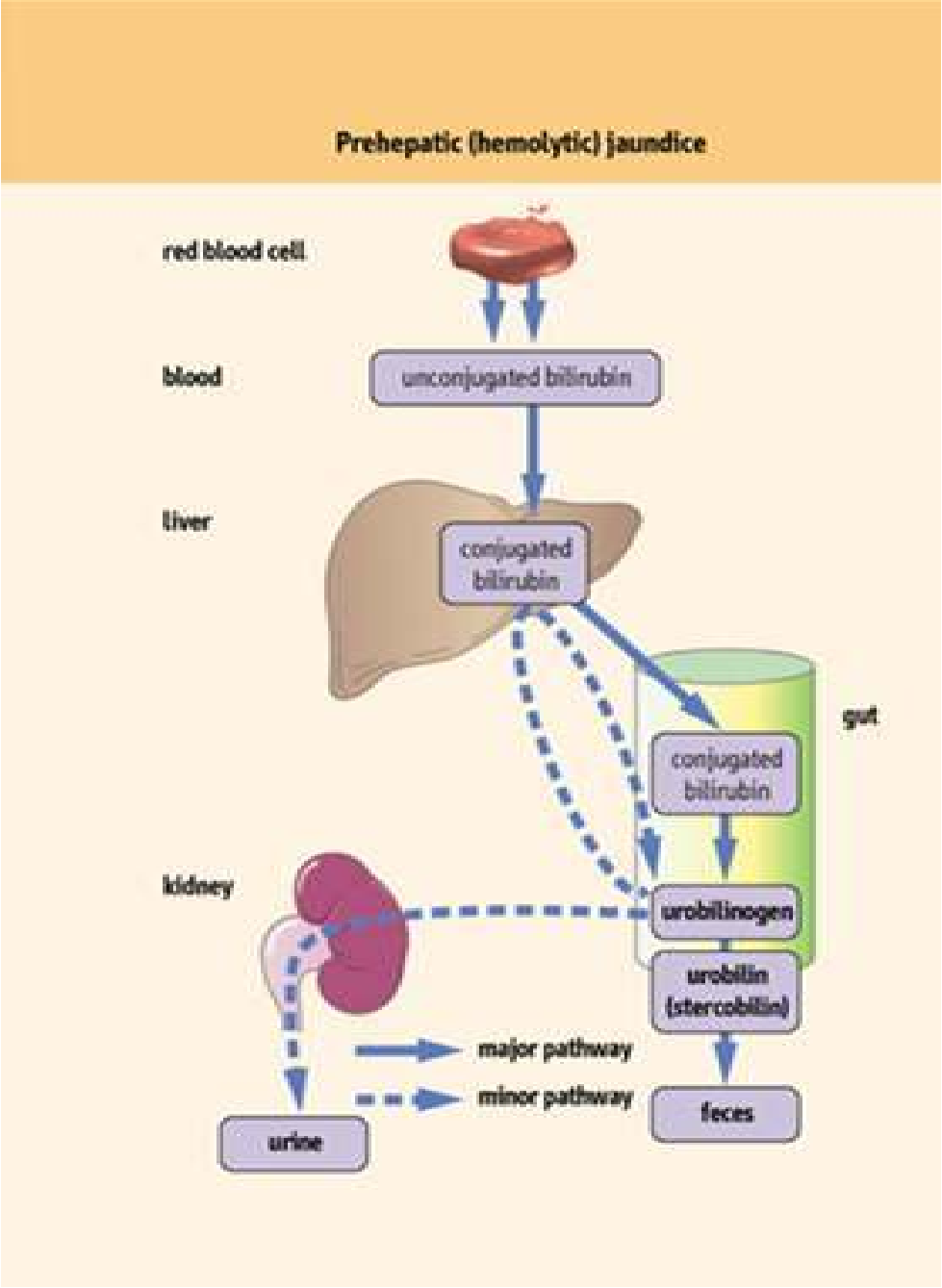


Fig. 5. Prehepatic Jaundice

HEPATIC JAUNDICE

As a result of liver dysfunction there is impairment of uptake , conjugation and secretion of bilirubin , hence both direct and indirect bilirubin level increases.

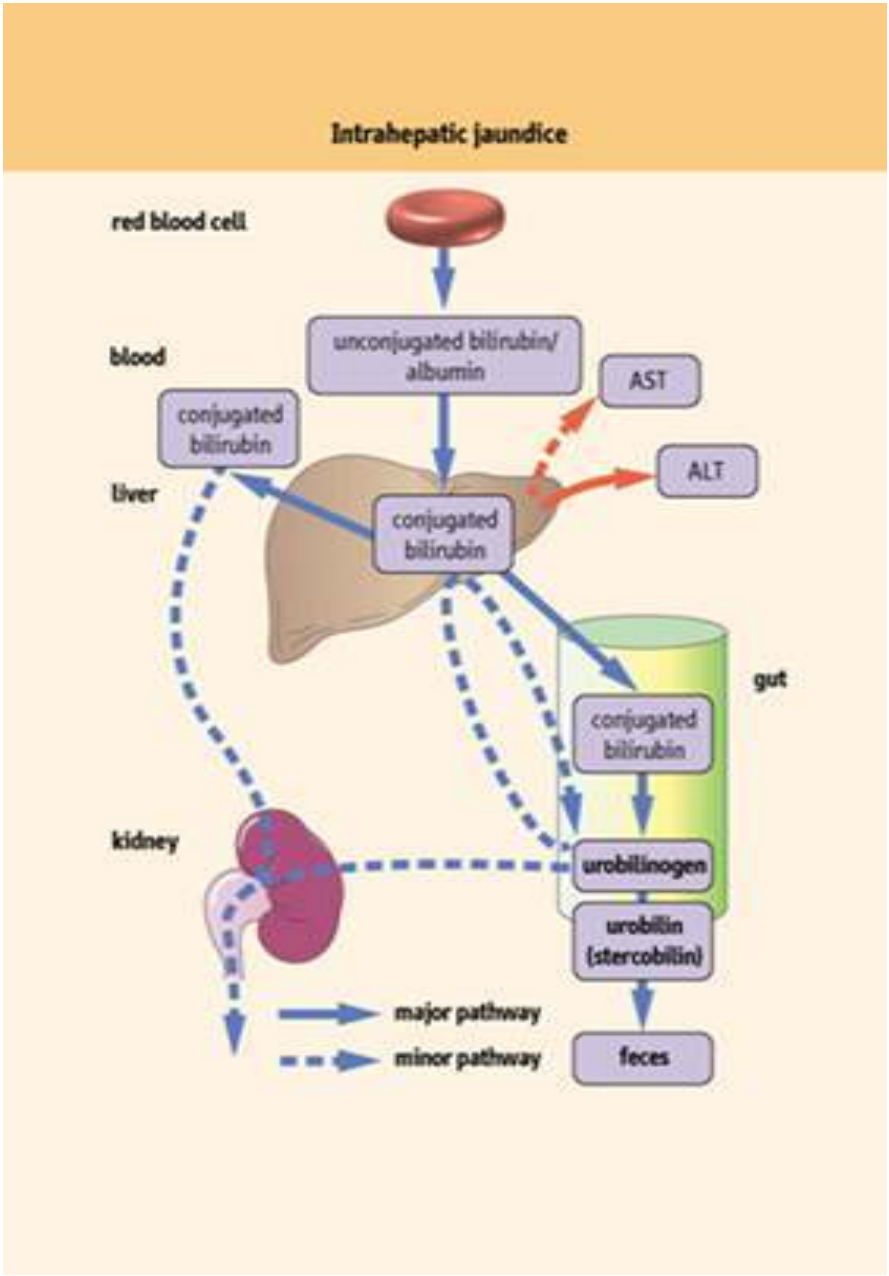


Fig. 6 Intrahepatic Jaundice

POSTHEPATIC JAUNDICE

When there is obstruction to biliary tree , the conjugated biliribin level gets elevated.

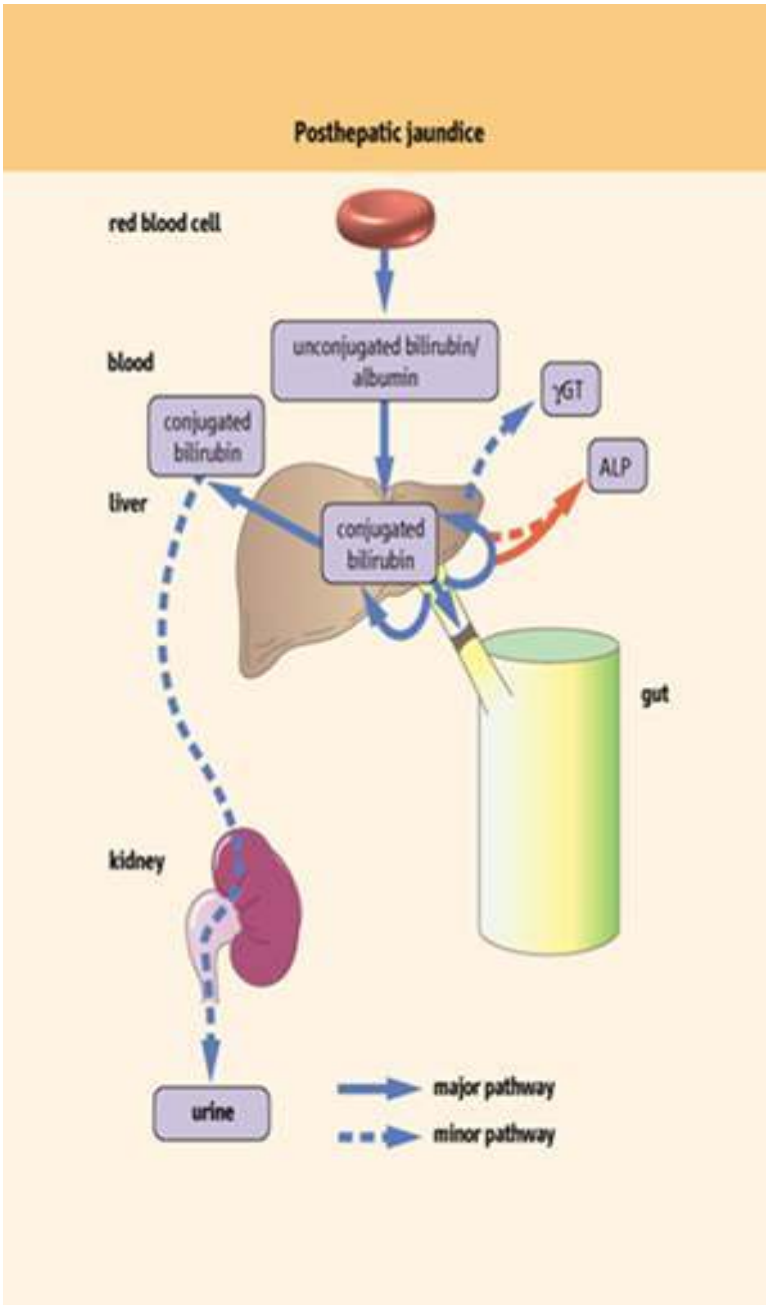


Fig. 7. Posthepatic Jaundice

Causes	Jaundice	Bilirubin in serum	Bilirubin in urine	Ubg in urine	Ubg in faeces
Prehepatic	Haemolytic	↑ indirect	No	↑	↑
Hepatic	Hepatic	↑Both direct and indirect	Yes	↓	↓
Posthepatic	Obstructive	↑Direct	Yes	No	No

REVIEW OF LITERATURE

In pregnancy the liver exhibits a range of altered functions including changes in blood flow to liver, cholesterol synthesis and secretion, motility of gall bladder is reduced.

Abnormal liver function test occur is 3%-5% of all pregnancies.

There will be rise in serum bilirubin level during pregnancy due to delayed excretion. A rise in alkaline phosphates, LDH and transaminases also noted. The rise in alkaline phosphatases is due to the contribution from placenta.

Hematocrit, serum urea, uric acid, albumin and total protein values falls,a rise is noted in cholesterol and triglyceride levels.

LIVER FUNCTION TEST & PREGNANCY

	Non-Pregnant	Pregnant
Bilirubin	2-17 μmol/L	No Change
Transaminases	7-40 IU/L	No Change
Gamma glutamyl transferase	<30 IU/L	No Change
Sr.Alkaline phosphatase	30-130 IU/L	Progressive rise after 5th week due to placental and skeletal component
Prothrombin time	8-14 sec	No Change
Total proteins	6.5-8mg/dl	decreases by 1mg/dl by 16th - 20th week
Albumin	3.5-5.5mg\dl	decreases by 1mg/dl mostly in first trimester
Globulin	3-5 mg/dl	Rises progressively to term
Fibrinogen	2-4 g/L	Rises progressively to term

CLASSIFICATION OF JAUNDICE IN PREGNANCY

I) Diseases unique to pregnancy

1) Hyperemesis gravidarum
2) Intrahepatic cholestasis of pregnancy
3) Pre-eclampsia
4) HELLP Syndrome
5) Acute fatty liver of pregnancy

II) Co-incidental to pregnancy

1) Viral Hepatitis
2) Gall stones
3) Drugs
4) Sepsis

III) Underlying chronic liver disease

1) Chronic hepatitis B & C
2) Autoimmune Hepatitis
3) Cirrhosis of liver
4) Wilson disease

HYPEREMESIS GRAVIDARUM

Intractable severe vomiting in the first trimester of pregnancy necessitating intravenous rehydration. Occurs in 0.3% of all pregnancies.

Etiology is hormonal, immunological and psychological factors. Risk factors include hyperthyroidism, psychiatric illness, molar pregnancy, pre existing diabetes and multiple pregnancies[1].

Liver dysfunction occur in 50% of patients with elevation in amino transferase upto 20 fold.

The balance of liver enzymes will return to normal when dehydration and malnutrition are corrected.

Both steroids and ondansetron are effective for treatment[2].

Rare complications include wernicke's encephalopathy, oesophageal rupture, central pontine myelinolysis, retinal haemorrhage[2].

INTRAHEPATIC CHOLESTATIS OF PREGNANCY

Second cause next to viral hepatitis for jaundice in pregnant woman. Occurs in second half of pregnancy with pruritis, elevated bile acid levels, disappears after delivery and recurrence is common.

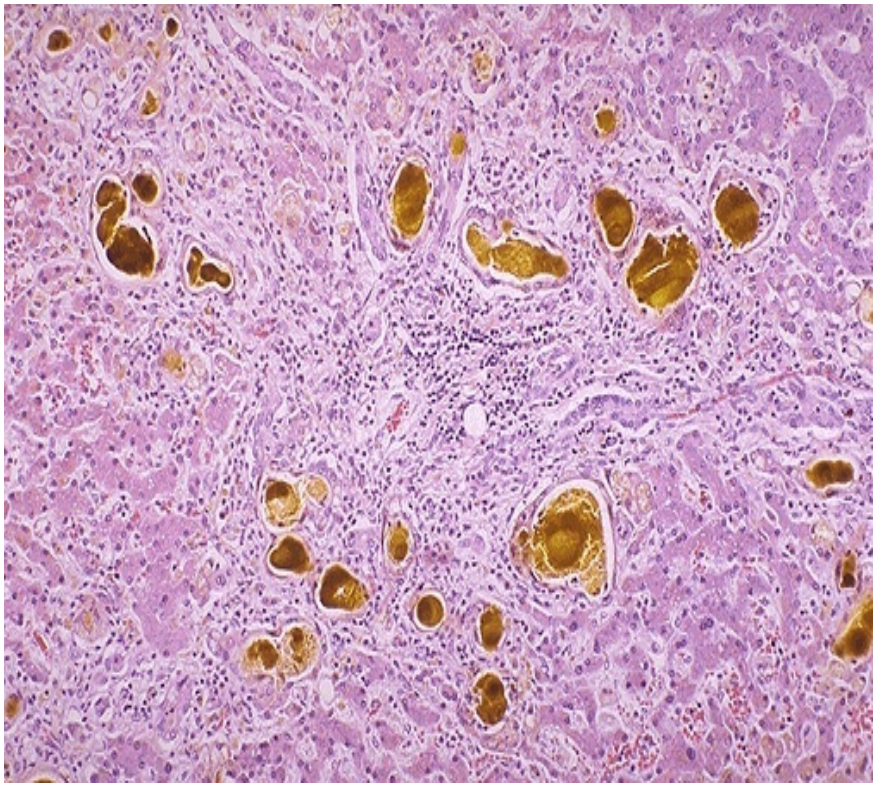


Fig. 8. Histopathology of Cholestasis Of Liver

IHCP is due to abnormal biliary transport across canalicular membrane the etiology for which is heterogenous with hormonal, genetic and exogenous factors exerting influence on hepatocyte or canalicular membrane of different individual.

Sex hormones have known cholestatic effects and abnormal progesterone metabolism is found in IHCP.

Atleast 10 different MDR3 mutations have been identified in ICP

One report has shown that certain MDR3 gene variants (ABCB4) is associated with severe form of IHCP[3].

Fetal complications such as sudden fetal death and premature labour are due to placental insufficiency and elevated fetal levels of bile acids[4].

High maternal levels of bile acids correlate with fetal morbidity and mortality[6].

CLINICAL FEATURES AND DIAGNOSIS

Pruritis starts around 25-32 weeks of gestation. It is worse at night and affects all parts of body. Jaundice occurs in 10-25% about 2-4 weeks after pruritis ,mild elevation to 10-20 fold elevation of amino transferase seen. Bilirubin is usually less than 5 mg/dl.

The most specific and sensitive marker of IHCP is serum bile acid. Levels of greater than 10μmol/l and sometimes upto 100 fold elevation is seen.

MANAGEMENT

- UDCA 10-15 mg/kg is treatment of choice [7]
- Dexamethasone 12 mg/day for 7 days.
- S.adenosyl methionine is less effective than UDCA but has an additive effect[8].

The main risk of IHCP is to the fetus. A swedish population study of more than 45,000 pregnancies with 693 cases of IHCP showed a positive correlation between maternal bile acid levels and premature delivery asphyxial events and meconium staining[6].

A recent large finnish population study showed that patients who have had IHCP developed cholecystitis, gall stones, non alcoholic pancreatitis, Hepatitis C and non alcoholic cirrhosis.

ACUTE FATTY LIVER OF PREGNANCY

It is a sudden catastrophic illness occurring most exclusively in third trimester, where microvesicular fatty infiltration results in encephalopathy and hepatic failure[9]. Incidence is approximately 1 in 14,000 pregnancies. Women with this condition give birth to male births commonly[10,11].

ETIOLOGY :

Etiology of acute fatty liver of pregnancy involves abnormalities in intra mitochondrial fatty acid oxidation[12]. Beta-oxidation of fatty acid in hepatic mitochondria requires several enzymes.

Mitochondrial trifunctional protein and its alpha subunit long chain 3-hydroxyacyl – CoA dehydrogenase (LCHAD) are 2 main enzymes and the genetic mutation in LCHAD is closely associated with AFLP.

LCHAD deficiency has been identified in about 20% of babies of mothers with AFLP. Maternal heterozygosity for LCHAD deficiency decreases the maternal capacity to oxidize long chain fatty acids in liver and placenta leading to accumulation of hepatotoxic LCHAD metabolites in maternal circulation[13].

CLINICAL FEATURES AND DIAGNOSIS

Burroughs et al., and others reported that women with AFLP have an increased frequently of Pre-eclampsia signs and are more likely in primigravida (50%) and have 10-15% increased incidence in twin pregnancies[14].

The presentation varies from asymptomatic to fulminant hepatic failure. Typical features would be 1 to 2 weeks of anorexia, headache, nausea, malaise, right upper quadrant pain with signs of jaundice, hypertension, ascites, small liver and hepatic encephalopathy 50% patients with AFLP have pre eclampsia and there is some overlap with HELLP syndrome[15].

Laboratorical features would be elevated bilirubin levels, amino transferase vary from near normal to 1000, usually about 300 to 500, normocytic normochromic anemia, high WBC count, normal to low platelets, coagulopathy with or without DIC, renal dysfunction, hypoglycemia.

Plasma urea, uric acid and creatinine levels are usually elevated. Uric acid levels may increase days before symptoms of AFLP become manifest[16], therefore serve as a pointer towards early diagnosis[17].

AFLP diagnosed presumptively by clinical and laboratorical features, a definitive diagnosis is histological Microvesicular fatty infiltration in zone 3 and portal inflammation with cholestasis seen.

Differential diagnosis include fulminant viral hepatitis and HELLP syndrome.

MANAGEMENT

Immediate termination of pregnancy and intensive supportive care is essential for both maternal and fetal survival.

INR of 1.5 and platelet count of >50,000 should be maintained, prophylactic antibiotic are recommended to prevent uterine infection.

With advanced supportive management the maternal mortality now is 7%-18% and fetal mortality 9%-23%

Infections and bleeding complications remain the most life threatening.

RECURRENCE AND SCREENING

Women who are carriers of LCHAD mutation have an increased risk of recurrence of AFLP in 20%-70% of pregnancies. Babies of mothers with AFLP should be screened for FAO defects as early diagnosis and appropriate management will reduce morbidity and mortality in these babies[18,19].

HYPERTENSION ASSOCIATED LIVER DYSFUNCTION OF PREGNANCY

Pre eclampsia :

Pre-eclampsia comprises a triad of Hypertension, edema and proteinuria, arises in third trimester and occurs in 5-10% of pregnancies.

With mild hypertension, 24% of patients have abnormal amino transferase levels, increasing to more than 80% in those with severe hypertension[20].

Liver involvement although infrequent always indicate severe pre eclampsia with significant morbidity and mortality.

It is the commonest cause of hepatic tenderness and liver dysfunction in pregnancy.

Early delivery is the key for treatment as hepatic complications accounts for 16% deaths of pre eclampsia, usually by hepatic rupture[21].

Periportal haemorrhage occurs in severe cases of eclampsia and in very severe cases they may form hematoma which ruptures [22,23] liver infarcts secondary to hepatic arterial thrombosis, which is associated with very high amino transferase levels.

The results of liver function test follow a standard pattern after delivery.

- Amino transferase level decrease after 24 h
- Bilirubin levels falls within 72 hrs.
- Alkaline phosphatase and GGT rise on day 5 or 6, peak on day 10 and return to normal within 8 weeks[24].

HELLP Syndrome

Severe pre eclampsia in 2%-12% of cases will be complicated by Hemolysis (H), Elevated liver enzymes (EL) and low platelet count (LP) the HELLP syndrome.

Diagnostic criteria for HELLP syndrome.

Hemolysis	Elevated liver tests	Low platelets
Abnormal blood smear	AST > 70/L	< 1,50,000
LDH> 600 U/L		
Increased Indirect Bilirubin		

HELLP syndrome may be subdivided, based on platelet count, into severe / class 1 (platelets < 50,000) Moderate / Class 2 (50-99,000) and Mild / Class 3(100-1,50,000)

ETIOLOGY :

HELLP syndrome is a microangiopathic hemolytic anemia associated with vascular endothelial injury leading to platelet consumption, resulting in small to diffuse haemorragic areas which spread to cause hematomas, capsular tears and intra peritoneal bleeding.

It was found that women heterozygous for factor V Leiden have an increased risk of developing HELLP syndrome[25] , while placental CD 95 ligand has been shown to act systematically to cause liver damage in patients with HELLP syndrome[26].

CLINICAL FEATURES AND DIAGNOSIS

Upper abdominal pain and tenderness, nausea, vomiting, malaise, head ache, edema and weight gain, Hypertension and proteinuria. Jaundice is uncommon (5%)

Common in older, white, multiparous ladies. Diagnosis is made on 3 laboratorical criteria (1) Hemolysis (2) Elevated aminotransferase (3) thrombocytopenia.

There is some overlap between AFLP and HELLP syndrome. HUS & TTP also have similar presentations.

MANAGEMENT

Majority of patients recover soon after delivery, only life threatening complications are taken as indication for specific therapy with plasmapheresis, plasma volume expansion, anti thrombotic agents, steroids, fresh frozen plasma dialysis.

Perinatal mortality is 11% due to prematurity, dysmaturity due to placental insufficiency.

RECURRENCE :

Pre eclampsia, recurrent HELLP, pre maturity, IUGR, abruption placentae recur in subsequent pregnancies in patients with HELLP syndrome.

VIRAL HEPATITIS IN PREGNANCY

Viral hepatitis is caused mostly by 5 hepatotrophic viruses, namely Hepatitis A, B, C, D, E.

Cytomegalovirus, Ebstein – Barr virus, Herpes simplex virus are rare causes of actue hepatitis in pregnancy.

Acute viral Hepatitis is characterized by marked increase in serum levels of ALT and AST due to liver cell necrosis. The serum level of alkaline phosphatase remains normal or mildly elevated, except for marked rise in infection due to hepatitis A virus or hepatitis E virus.

Hepatitis A virus

Non enveloped 27 nm RNA virus, transmitted through faeco oral contamination.

Poor hygiene, poor sanitation and intimate personal or sexual contact facilitate transmission.

In developing countries, more than 90% population is exposed to HAV infection in childhood and possess anti-HAV antibody

Acute hepatitis A is a self limiting disease and the prognosis in pregnancy is the same as non pregnant patient.

Prevention is by avoidance of exposure and vaccination.

Hepatitis B Virus

Double standard DNA virus.

Genome of HBV has four open reading frames that encode four major proteins surface protein (HBSAG), Core proteins (HBC Ag & HBE Ag), DNA polymerase and X protein.

Once a person gets infected with HBV, first viral marker detected is serum HbSAg followed by elevation of serum transaminases and clinical symptoms.

HbSAg becomes undetectable 1 or 2 months after the onset of jaundice and rarely persists after 6 months. Afterwhich, anti HbSAg becomes detectable in serum.

HBC Ag confined to liver cells and therefore not detectable in serum.

Anti HBC is readily demonstrable in serum 1 or 2 weeks after appearance of HbSAg.

Recent and Remote infection distinguished by Immunoglobulin class of anti HBC. IgM anti-HBC predominates in first 6 months whereas beyond 6 months Ig G – anti HBC predominates.

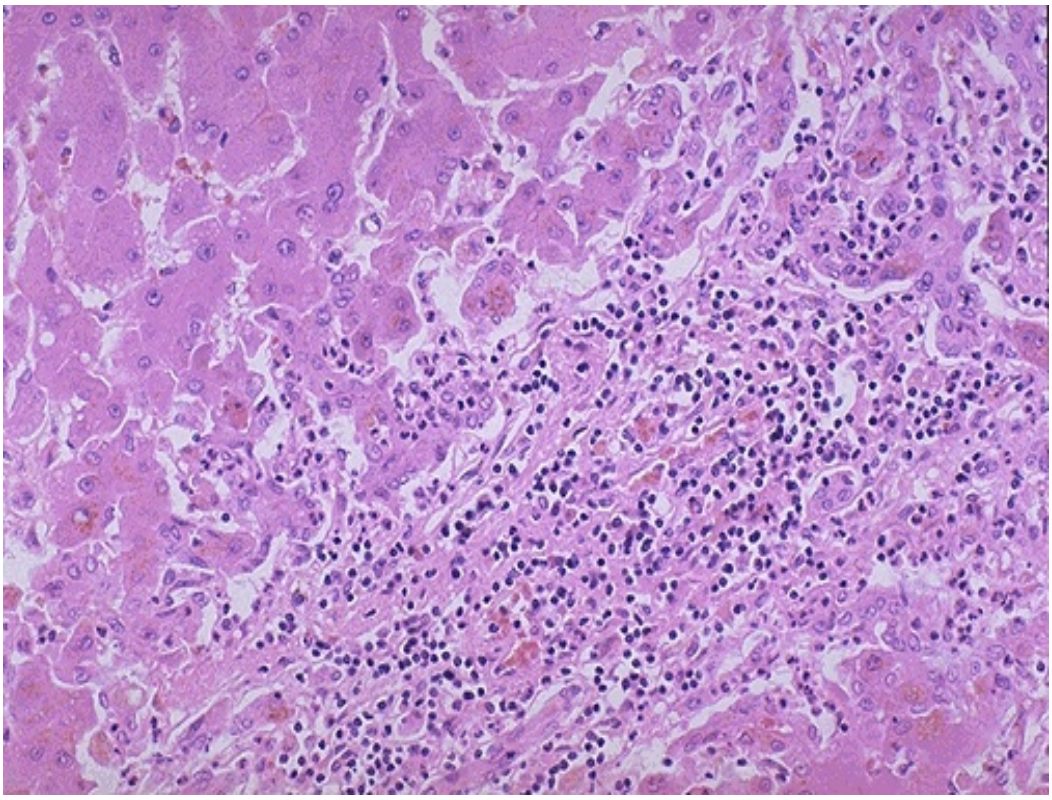


Fig. 9. Histopathology of Acute Viral Hepatitis

Hbe Ag becomes detectable in serum concurrent with or short after appearance of HbSAg.

Earlier the age of acquisition of HBV infection, higher the propensity for chronic disease. The natural course of HBV in chronic carriers varies from an inactive state to recurrent acute exacerbations. Chronic hepatitis, cirrhosis and hepatocellular carcinoma may develop after a prolonged period.

When an asymptomatic pregnant woman is found to be hepatitis B surface antigen positive at the time of her first antenatal visit, we have to differentiate between a chronic carrier and an acute infection.

No evidence proves that hepatitis B infection is more common during pregnancy. As HBe Ag is a marker of active replication. If the mother is positive for both HBs Ag and HBe Ag, the risk of transmission to child is 70-90%, whereas the risk of transmission is 10-20% if mother positive only for HBs Ag.

Infants of HBsAg positive mothers should receive Hepatitis B immunoglobulin (HBIG) in addition to the HBV vaccine. HBIG when given to newborn in first 24 hours of delivery infection is prevented in 80-85%

HEPATITIS C VIRUS

Enveloped single stranded RNA virus. Only 10-15% of HCV infected patients develop hepatitis and the rest remain asymptomatic. 50-85% become chronic carriers. Process of liver damage is slow and takes about 30-40 years before cirrhosis and hepato cellular carcinoma occur.

There may be intermittent or persistent risk in serum transaminases accompanied by histological changes in the liver that range from a mild to severe chronic active hepatitis and cirrhosis.

Mode of transmission is via blood and blood products, sexual contact and sharing of needles among intravenous drug abusers.

Maternal – infant transmission has become an important mode of spread of infection.

Co-infection with HIV increases the risk for HCV transmission significantly.

Type of delivery does not seem to influence the rate of mother to infant transmission.

Routine screening for her in pregnancy not recommended, However, screening of high risk groups, such as HIV patients, previous or current injectable drug abuse, those who received blood transfusions before becomes mandatory.

Hepatitis D Virus

Single stranded circular RNA virus, which depends on the presence of HBV for its replication. Co-infection with HBV and HDV causes more severe disease with higher chronicity rate.

Hepatitis E Virus

Non-enveloped, single stranded RNA virus.

Diagnosis of acute hepatitis E infection is made by demonstration of IgM anti HEV antibodies in the serum.

Pregnant women, particularly those in second and third trimesters, are more frequently affected during Hepatitis E outbreaks.

Disease is more severe when infection occurs in third trimester with mortality rates between 15% to 25%

HEV infection adversely affects fetal outcome, with increased incidence of abortions, still births and neonatal deaths.

Administration of immunoglobulins to pregnant women during on outbreak of HEV has been reported to reduce the risk of infection.

Gall Stones :

Cholesterol secretion increases in the second and third trimester compared to bile acids and phospholipids, leading to supersaturated bile, in addition, fasting and post prandial gall bladder volumes are greater with reduced rate and volume of emptying.

Upto 10% of patients develops stone on sludge over the course of one pregnancy with obesity and serum leptin being risk factors[28].

Commonest clinical presentation is Biliary colic (5% jaundice in pregnancy), gall stone pancreatitis and least commonly acute cholecystitis.

Biliary colic will recur in 50% of pregnant patients. Before delivery, symptomatic patients should undergo laparoscopic cholecystectomy in the second trimester with better pregnancy outcome compared with medical therapy[29,30].

UNDERLYING CHRONIC LIVER DISEASES

CIRRHOSIS AND PORTAL HYPERTENSION

Most patients with advanced cirrhosis are amenorrhic and infertile. Successful pregnancy may be completed in those with well compensated disease and only mild portal hypertension. Increased maternal and fetal problems can be expected in about 50% cases with increased fetal loss and maternal risks of hepatic decompensation, jaundice, thrombocytopenia, rupture of splenic aneurysm, variceal bleeding (20-25%). Patients with oesophageal varices should be considered for endoscopic therapy, shunt surgery or even liver transplantation before pregnancy.

Fig. 10 Caput Medussae

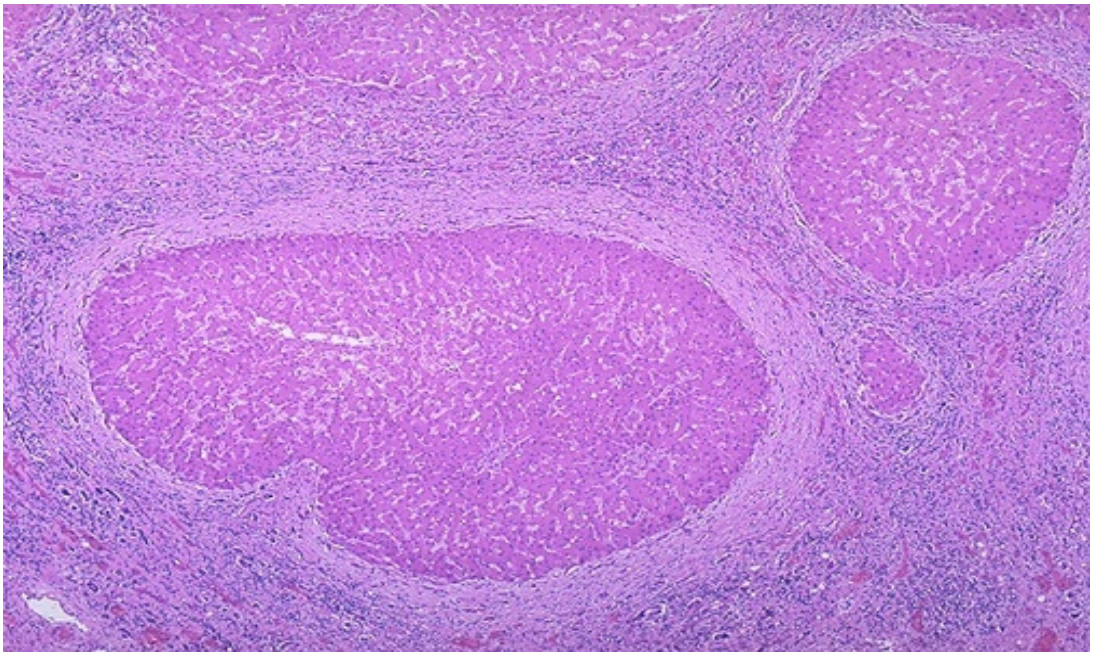


Fig. 11 Histopathology of Cirrhosis Of Liver

Vaginal deliveries with assisted second stage of labour can be done in mild cases. But with large varices, avoidance of labour by cesarean section is recommended to avoid increase in portal pressure and risk of variceal bleeding.

Correction of coagulopathy and prophylactic antibiotics given to reduce the incidence of postpartum haemorrhage and bacterial infection.

OTHER INFECTIONS

Malaria :

Malarial infection can be endemic or sporadic caused by Plasmodium species. The incubation period varies from 8-25 days. The anopheles mosquito is the vector. Symptoms are fever and flu like symptoms including malaise, vomiting and headache which may occur in intervals. Symptoms are less severe in immune patients. Malaria may be associated with jaundice and anaemia. Jaundice is due to hemolysis and hepatitis. Liver and spleen enlarge and become tender.

Effect on pregnancy :

Pregnant women have an increased incidence of infection .Parasitaemia may be great. Severe anaemia in pregnancy is a major obstetric problem in malaria endemic area. Haemolytic anemia which can cause jaundice may be severe in pregnancy and hepatorenal syndrome is often the cause of death. Malaria may present as puerperal pyrexia following delivery in women with low immunity.Most important complication is Postpartum haemorrhage. Malaria can infect placenta leading to maternal anaemia, spontaneous abortion, intrauterine death, stillbirth and low birth weight. Intrauterine death is caused by massive infection of the placenta and persistent high fever.In non-immune

mothers malarial parasites can cross placenta leading to congenital malaria. In endemic areas WHO recommends malarial chemoprophylaxis throughout pregnancy. It is given as weekly prophylaxis with chloroquine in a dose of 300mg/wk.

LEPTOSPIROSIS :

About 5-10% of patients with leptospirosis will have hepatic and renal involvement. Transmitted by direct contact with urine, tissue or blood of an infected animal. Entry is through abrasions in skin or through intact mucous membrane. Incubation period is 2-20 days. 90%of patients have relatively mild and anicteric form of leptospirosis. Severe leptospirosis may present as renal dysfunction, jaundice and haemorrhagic diathesis referred as Weil's syndrome. There is marked elevation of serum billirubin and alkaline phosphatase with moderate elevation of aminotransferases in patients with liver involvement. Diagnosis is based on either isolation of the organism or a rise in antibody titre in Microscopic slide agglutination test (MAT).

Effect on pregnancy :

Perinatal mortality is high in leptospirosis. 30% of pregnancies ended in abortion or preterm death**.** Vertical transmission is rare. Drug of choice is Penicillin G in a dose of 1.5million units IV qid. Other

alternative are erythromycin and ampicillin. Doxycycline is contraindicated in pregnancy.

Typhoid :

Enteric fever is endemic in most developing regions especially in India. Transmission is by ingestion of contaminated water or food. Incubation period is 3-21 days. The prominent symptom is prolonged fever. It is seen in 75% of cases. Gastrointestinal symptoms are quite variable. Only 20-40% of patients have abdominal pain. Hepatic dysfunction, though less common can occur in patients with typhoid fever. Moderate elevation of Liver function tests might be present.

Effects on Pregnancy :

The acute infection in pregnant women run a similar course to those in non pregnant women. Vertical transmission has been recorded**.** Around 3% of patients with severe form of typhoid fever may go for spontaneous abortions and preterm labor. But effective antibiotic treatment has made this outcome less common. Chloramphenicol is the drug of choice but other options are ciprofloxacin, azithromycin and ceftriaxone.

Herpes Viruses :

All members of herpes group – Herpes simplex, Epstein barrvirus, cytomegalo virus and varicella can cause hepatitis. More than half are associated with immunosuppression. Jaundice is not invariable and mucocutaneous stigmata may be absent. Herpes simplex hepatitis carries a grave prognosis. Mortality exceeds 90% even with treatment. Herpes simplex type II is most common in 3rd trimester. Symptoms are three times more common in pregnancy. The virus can cross placenta but it has no relation with congenital malformation. HSV II acquired at birth from cervical and vaginal contamination can lead to disseminated infection in babies with 90% mortality. Pregnancy is a high risk factor and has been associated with a high maternal and fetal mortality rate. Acyclovir appears to improve the outcome.

Congenital non-haemolytic Hyperbilirubinemia :

GILBERT'SDISEASE:

Gilberts disease presents as mild fluctuating jaundice which may only be noticed when the patient is tired, dehydrated or calorie depleted. As all of these may occur during normal pregnancy, diagnosis of gilbert's disease may be made for the first time during pregnancy. Serum billirubin is elevated, but liver enzymes are normal, there is no sign of any systemic

disease and no symptoms other than jaundice. Maternal and perinatal outcome are not affected by the disease. It is relatively common in Western Societies with the prevalence rate of 1-2%. There are no significant risks with the diagnosis but precipitating factors should be minimized.

MATERIALS AND METHODS

Forty Six women with jaundice complicating pregnancy admitted and treated at Government Coimbatore Medical College Hospital, Coimbatore from August 2013 to August 2014 were studied.

- A detailed history including patient's age, socioeconomic status, booking, parity and details of menstrual history to arrive at the expected date of delivery was obtained.

- Patients were enquired in detail about their complaints and duration like nausea, vomiting, pruritus, anorexia, yellow coloured urine, pale stools, edema legs, bleeding tendency, joint pain, fever and others.

- Past history of jaundice especially in previous pregnancy and history of blood transfusion were elicited.

- Systemic and obstetric examinations were carried out.

- Investigations included liver function tests, serum billirubin, SGOT, SGPT, alkaline phosphatase, Viral markers, prothrombin time (PT), partial thromboplastin time (PTT), bleeding time (BT), clotting time (CT), platelet count and ultrasound abdomen were carried out as and when required.

- HIV screening was done in all patients.

- Medical gastroenterologist opinion was obtained for all cases.

- Labour was closely monitored. Jaundice per se was not an indication for cesarean section. Vaginal delivery with close monitoring was preferred and cesarean sections were done only for obstetric indication. After cross matching fresh blood, fresh frozen plasma was kept ready as alteration in coagulation profile was expected in jaundice complicating pregnancy.

- Atonicity was managed with oxytocin drip , injection methergin and injection 15 methyl PGF2α.

- Patient were kept in the labour ward for close observation. Clotting time was repeated hourly if it was prolonged till it becomes normal.

- Soon after delivery all babies were assessed by paediatrician. Alive or dead , sex , gestational age at birth , weight , apgar score and presence or absence of any congenital anomalies were looked for and noted. As per paediatrician opinion sick babies were admitted in preterm ward for intensive care.

- The maternal outcome was noted in terms of the mode of termination of pregnancy, maternal complications and maternal mortality.The relation of maternal morbidity and mortality to the admission serum bilirubin level was analysed.

- To identify the various etiologies and distribution of jaundice with reference to age , parity and trimesters.

- Fetal outcome was assessed by perinatal morbidity and mortality

- Study design: Prospective cohort study

- Study Population : 46 patients

- Inclusion Criteria : All jaundiced antenatal mothers attending Obstetrics and Gynaecology Department in Coimbatore Medical College Hospital.

- Exclusion Criteria : Patients on Hepatotoxic Drugs, Jaundice due to Sepsis.

OBSERVATIONS AND RESULT

The incidence of jaundice in India varies from 0.4-0.9 per 1000 deliveries. A prospective study of all antenatal patients admitted to Coimbatore medical college with jaundice was evaluated from august 2013- august 2014. Total number of admissions during this period was 12,286. According to this study the incidence of jaundice is 3/1000 deliveries.

Singh et al reported a jaundice incidence of 1.03/1000.kamala jayaram and rama devi 2 reported an incidence of 0.4/1000.

AGE GROUP:

Of the 46 women studied ,45.7% were in the age group of 21-25 years(TABLE 1)

PARITY AND GESTATIONAL AGE:

About 41.3% women of 46 were primi and 30.4% second gravida. The incidence is more common in third trimester 93.5 %(TABLE 2 & 3)

ETIOLOGY OF JAUNDICE:

Of the 46 women, 25 had viral hepatitis (54.3%), 12 had HELLP syndrome(26.1%),3 had acute fatty liver of pregnancy (6.5%), 3 had intra hepatic cholestasis of pregnancy (6.5%),cirrhosis (6.5%) (TABLE 7).

COMPLICATIONS :

Of the 46 women , 33 recovered without major complications (71.7%), 5 developed DIC (10.9%), 3 had Atonic PPH (6.5%), out of which 2 expired, 1 developed hepatic encephalopathy (2.2%), 1 had ARF (2.2%), 1 splenic rupture (2.2%),1 variceal bleed(2.2%), 1 vulval hematoma (2.2%). (TABLE 8)

MATERNAL OUTCOME:

Among the 46 women studied, 43 delivered and 3 were undelivered, of the 3 undelivered 2 expired, out of the 43 delivered ,23 was labour natural (50%),19 had LSCS (12.5%), 1 was VBAC (12.5%). Of the 8 deaths 3 were due to DIC (37.5%),2 due to Atonic PPH (25%),1 developed hepatic encephalopathy (12.5%),1 of splenic rupture (12.5%), 1 of variceal bleed (12.5%).(TABLE 9)

FETAL OUTCOME:

Of the 43 delivered, 37 was live birth (86%), 6 dead born (14%). Out of 37 , 28 were term babies (75.7%), 9 were preterm (24.3%). 20 male babies (46.5%),23 female babies (53.5%).44.2% of babies weighed <2.5 kg, 51.2% weighed between 2.5-3.5 kg, only 4.7% weighed more than 3.5 kg.(TABLE 12,13,14,15).

ANALYSIS

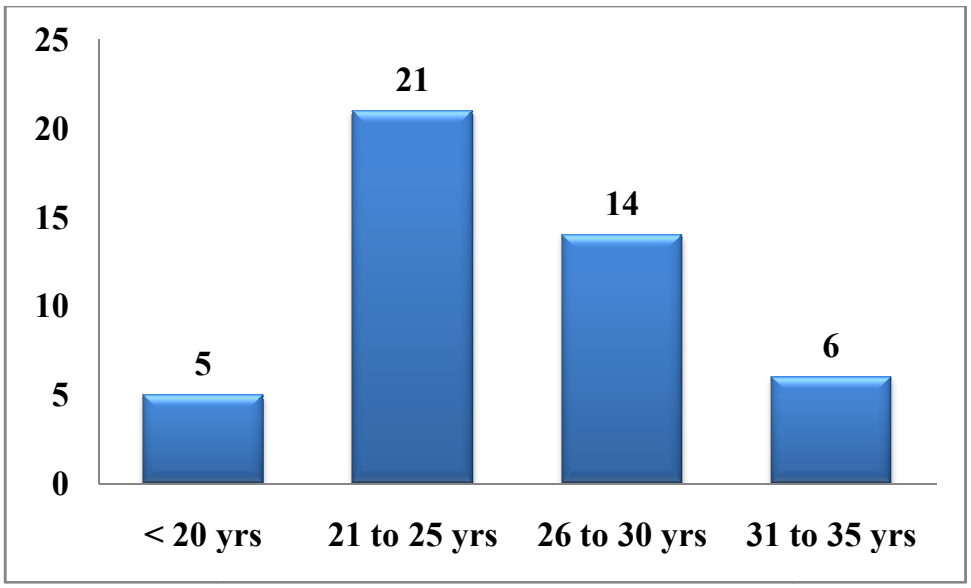
TABLE 1

AGE DISTRIBUTION

AGE	FREQUENCY	PERCENTAGE
<20	5	10.9
21-25	21	45.7
26-30	14	30.4
31-35	6	13
TOTAL	46	100

CHART 1

AGE DISTRIBUTION



Out of the 46 women studied , 45.7% were in the age group of 21-25 years.

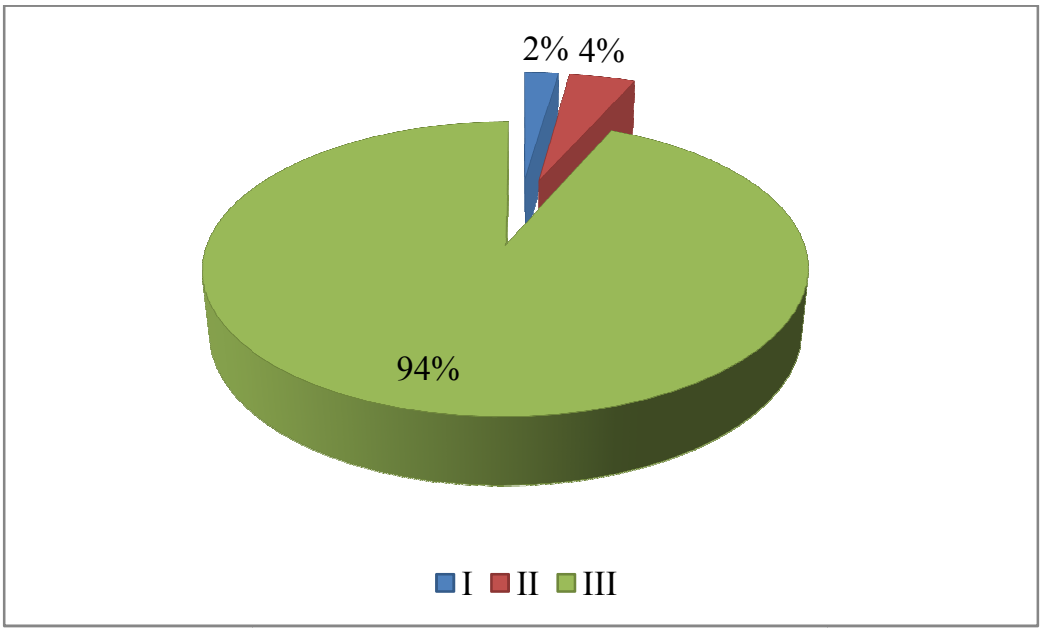
TABLE 2

TRIMESTER DISTRIBUTION

S.NO	TRIMESTER	FREQUENCY	PERCENTAGE
1	I	1	2.2
2	II	2	4.3
3	III	41	93.5
	TOTAL	44	100

CHART 2

TRIMESTER DISTRIBUTION



Jaundice frequency is high in third trimester with a percentage of 93.5.

TABLE 3

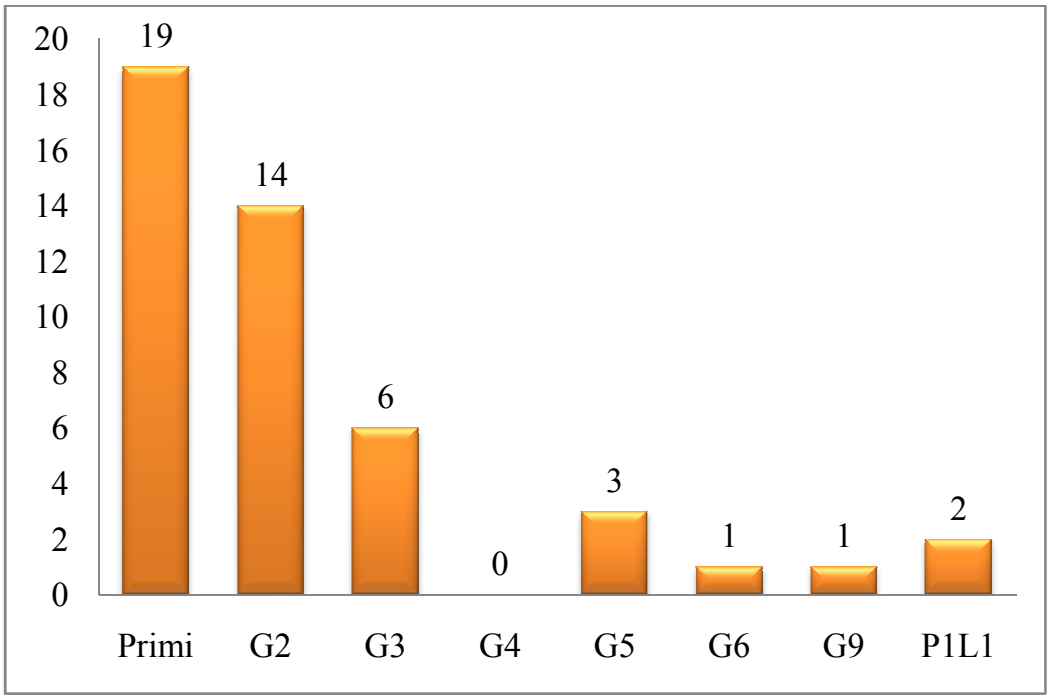
PARITY DISTRIBUTION

S.NO	PARITY	FREQUENCY	PERCENTAGE
1	Primi	19	41.3
2	G2	14	30.4
3	G3	6	13.0
4	G4	-	0.0
5	G5	3	6.5
6	G6	1	2.2
7	G9	1	2.2
8	P1L1	2	4.3
	TOTAL	46	100

Out of the total 46 woman, 41.3% were primi and 30.4% were second gravida.

CHART 3

PARITY DISTRIBUTION



Out of the total 46 woman, 19 were primi and 14 were second gravida.

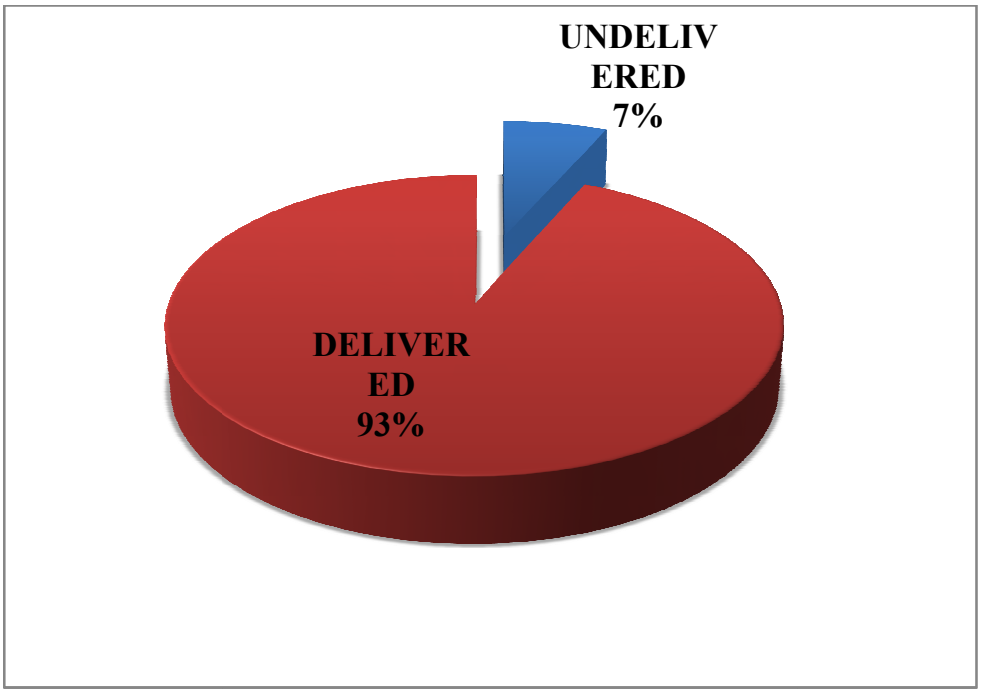
TABLE 4

PREGNANCY OUTCOME

S.NO	OUTCOME	TOTAL	PERCENTAGE
1	UNDELIVERED	3	6.5
2	DELIVERED	43	93.5
	TOTAL	**46**	**100**

CHART 4

PREGNANCY OUTCOME (TABLE 4)



Of the 46 women, 43 delivered and only 3 were undelivered, of the 3 undelivered 2 expired.

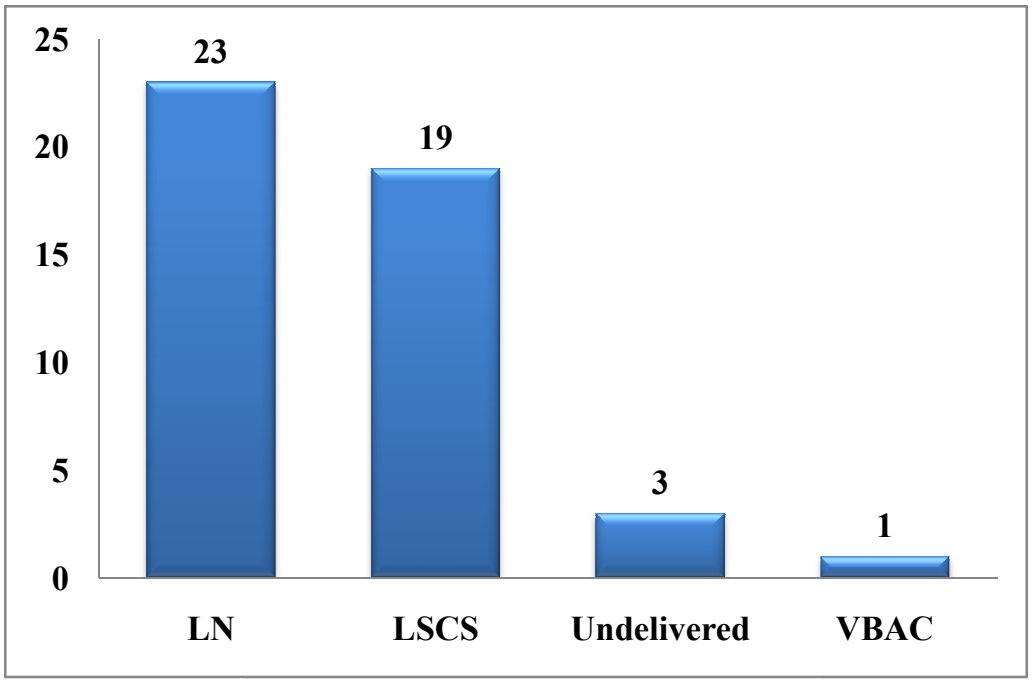
TABLE 5

MODE OF DELIVERY

S.NO	MODE OF DELIVERY	FREQUENCY	PERCENTAGE
1	LABOUR NATURALE	23	50
2	LSCS	19	41.3
3	UNDELIVERED	3	6.5
4	VBAC	1	2.2
	TOTAL	**46**	**100**

CHART 5

MODE OF DELIVERY



Of the 46 women , 43 delivered , among them 23 had labour natural, 19 underwent lscs, 1 had VBAC.

TABLE 6

LEVEL OF INITIAL BILIRUBIN

S.NO	INITIAL BILIRUBIN	TOTAL	PERCENTAGE
1	<5	25	54.3
2	5-10	10	21.7
3	10-15	5	10.9
4	>15	6	13

of the 46 women, 25 that is 54.3% had a initial bilirubin level of <5 , and only 6 women had bilirubin levels >15 , among them 3 expired giving that 50% of jaundiced patients with bilirubin levels more than 15 had mortality.

CHART 6

LEVEL OF INITIAL BILIRUBIN

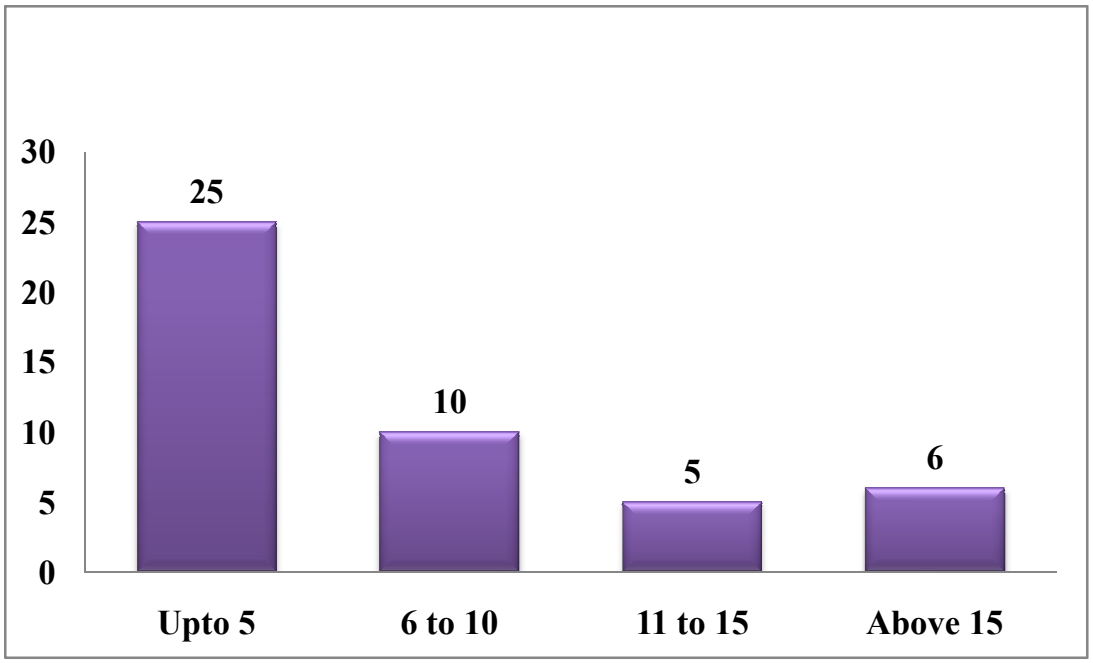


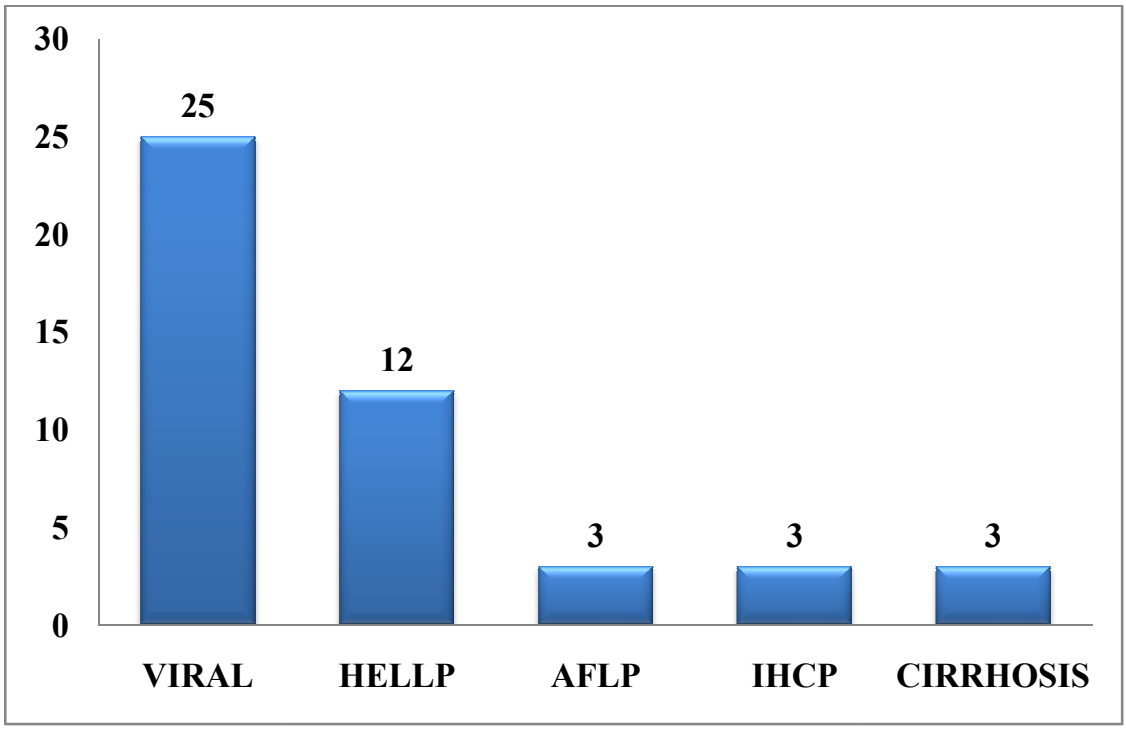
TABLE 7

ETIOLOGY OF JAUNDICE

S.NO	ETIOLOGY	TOTAL	PERCENTAGE
1	VIRAL	25	54.3
2	HELLP	12	26.1
3	AFLP	3	6.5
4	IHCP	3	6.5
5	CIRRHOSIS	3	6.5
6	TOTAL	46	100

CHART 7

ETIOLOGY OF JAUNDICE



Of the 46 studied, 25 had viral hepatitis (54.3%), 12 had HELLP syndrome(26.1%),3 AFLP,3 cholestatic jaundice and 3 cirrhosis with portal hypertension.

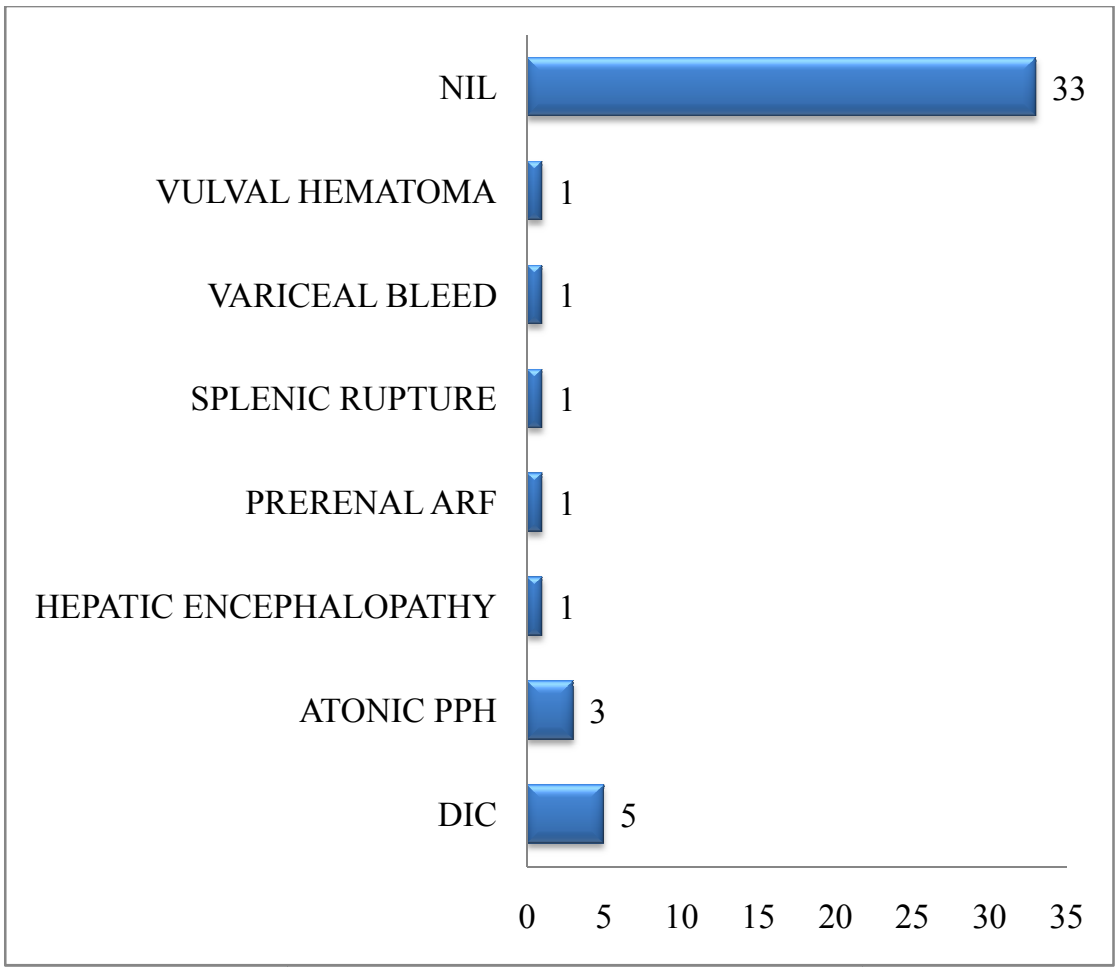
TABLE 8

MATERNAL COMPLICATIONS

S.NO	COMPLICATIONS	TOTAL	PERCENTAGE
1	DIC	5	10.9
2	ATONIC PPH	3	6.5
3	HEPATIC ENCEPHALOPATHY	1	2.2
4	PRERENAL ARF	1	2.2
5	SPLENIC RUPTURE	1	2.2
6	VARICEAL BLEED	1	2.2
7	VULVAL HEMATOMA	1	2.2
8	NIL	33	71.7
	TOTAL	**46**	**100**

CHART 8

MATERNAL COMPLICATIONS



Of the 46, 33 recovered without much complications, out of the 13 who developed complications ,5 had DIC, 3 atonic pph, each one had splenic rupture, hepatic encephalopathy, vulval hematoma, pre renal ARF, variceal bleed.

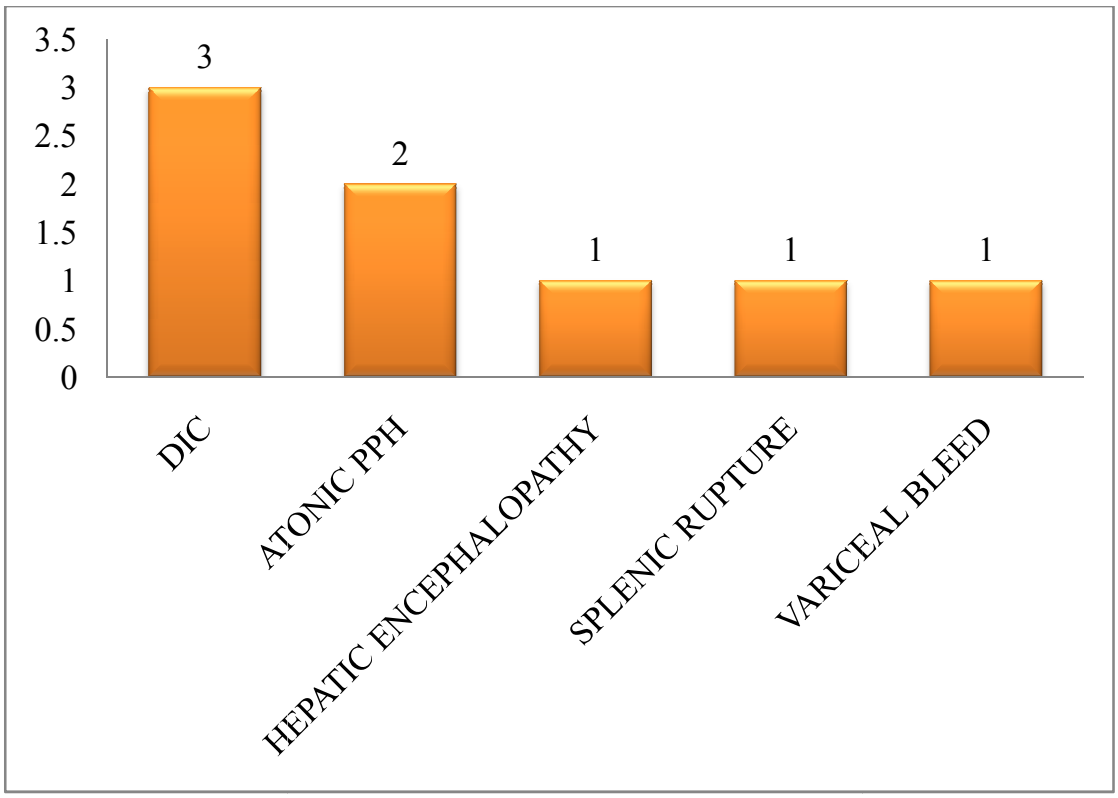
TABLE 9

CAUSE OF DEATH

S.NO	CAUSE OF DEATH	TOTAL	PERCENTAGE
1	DIC	**3**	**37.5**
2	ATONIC PPH	**2**	**25**
3	HEPATIC ENCEPHALOPATHY	**1**	**12.5**
4	SPLENIC RUPTURE	**1**	**12.5**
5	VARICEAL BLEED	**1**	**12.5**
	TOTAL	**8**	**100**

CHART 9

CAUSE OF DEATH



8 women expired, DIC as a cause in 3 (37.5%),atonic pph in 2 (25%),hepatic encephalopathy in 1 (12.5%), splenic rupture in 1 (12.5%), variceal bleed (12.5%).

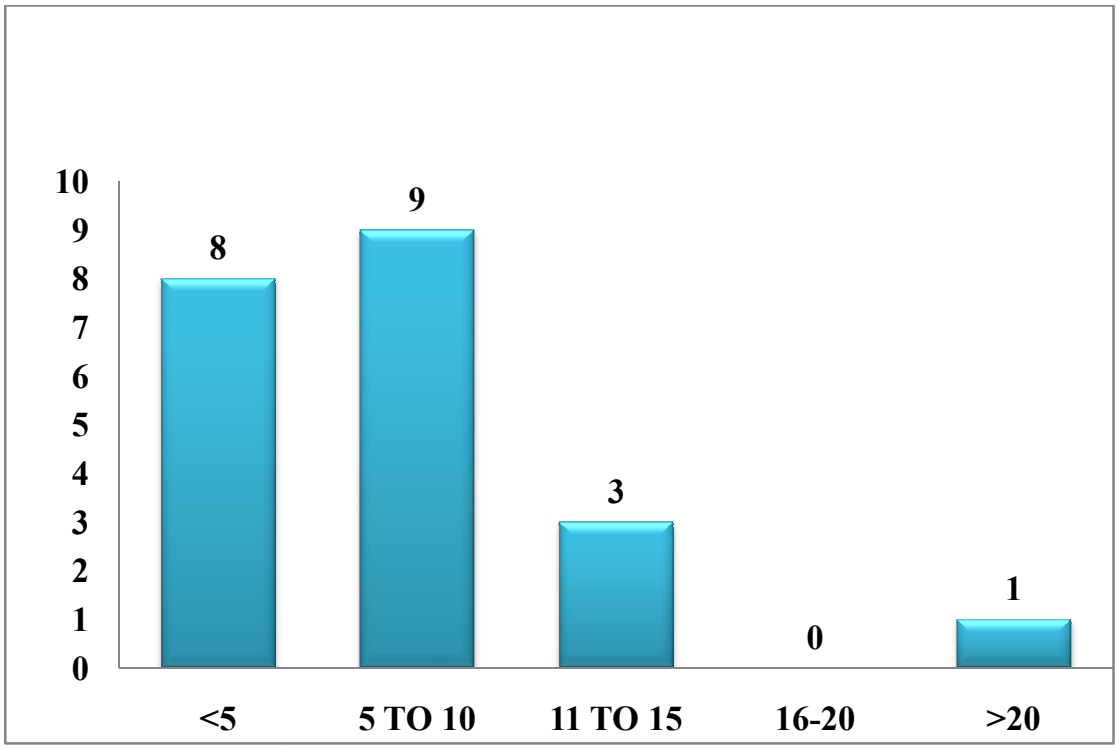
TABLE 10

BLOOD TRANSFUSIONS

S.NO	TRANSFUSIONS	TOTAL	PERCENTAGE
1	<5	8	38.1
2	5-10	9	42.9
3	11-15	3	14.3
4	16-20	-	0
5	>20	1	4.8

CHART 10

BLOOD TRANSFUSIONS



Of the 46 women, 21 had multiple transfusions,8 had <5 transfusions, 9 had 5-10 transfusions,and only one had >20 transfusions.

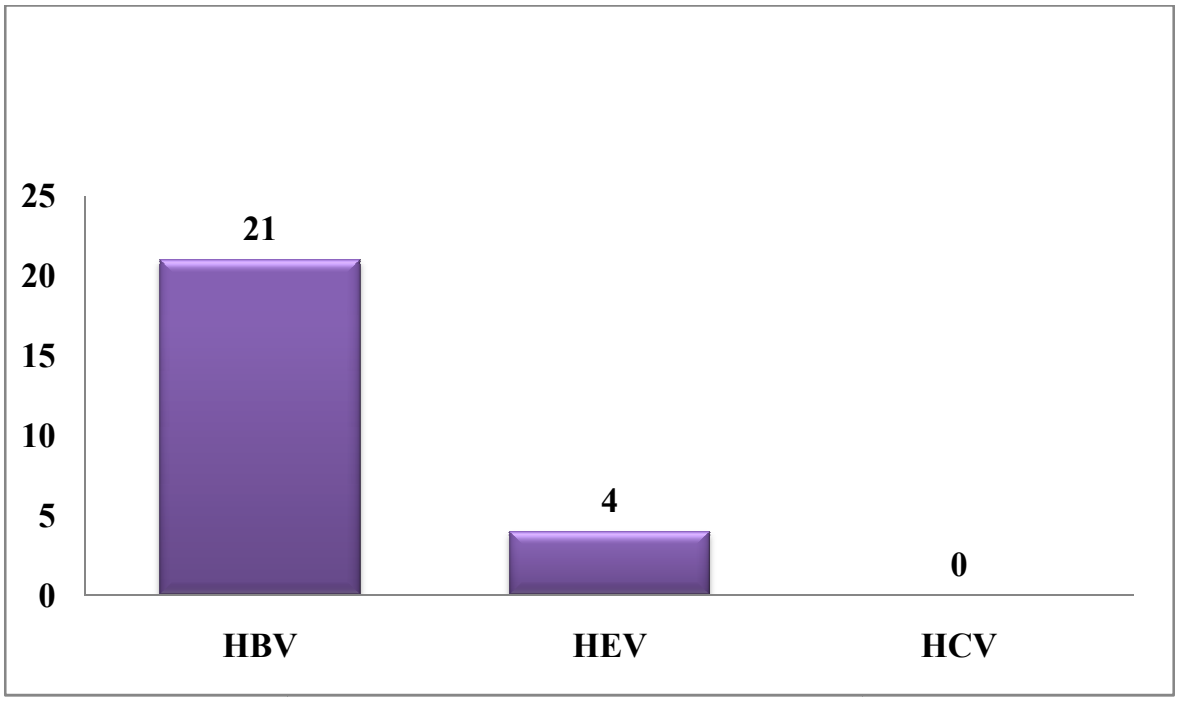
TABLE 11

VIRAL ETIOLOGY

S.NO	ETIOLOGY	TOTAL	PERCENTAGE
1	HBV	21	84
2	HEV	4	16
3	HCV	-	
	TOTAL	**25**	**100**

CHART 11

VIRAL ETIOLOGY



Hepatitis is the most common of cause of jaundice in pregnancy,out of 46, 25 had hepatitis, among which 21 had hepatitis B infection and 4 had hepatitis E.

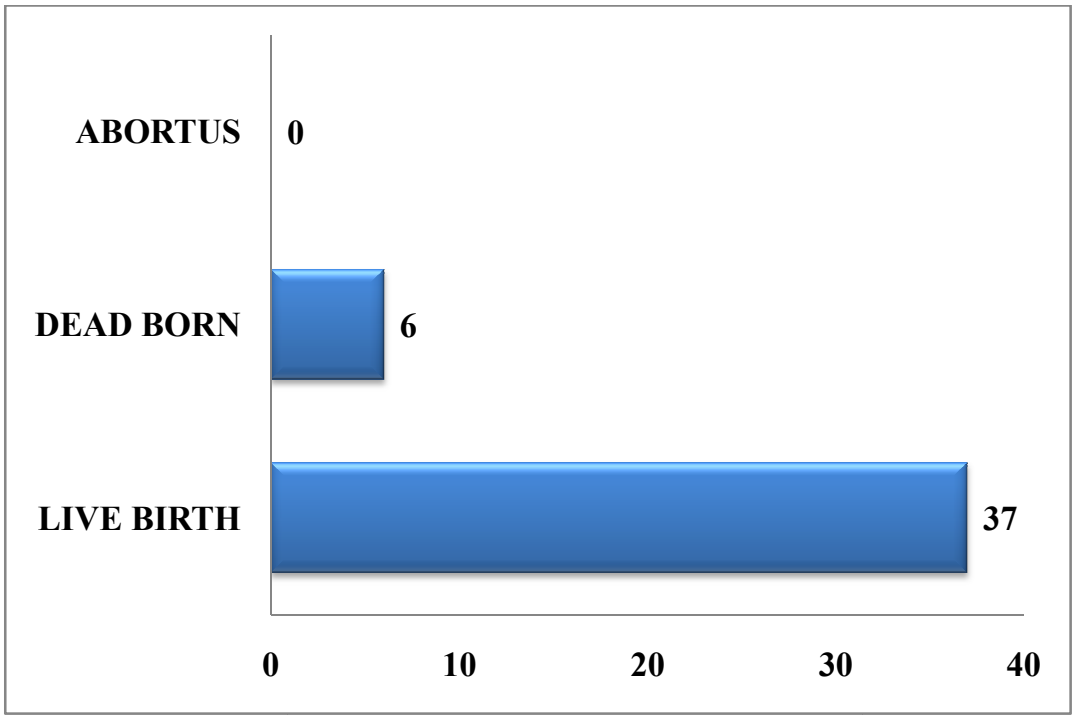
TABLE 12

FETAL OUTCOME

S.NO	OUTCOME	TOTAL	PERCENTAGE
1	LIVE BIRTH	37	86
2	DEAD BORN	6	14
3	ABORTUS	-	
	TOTAL	**43**	**100**

CHART 12

FETAL OUTCOME



of the 46, 43 delivered, 37(86%) were live births, 6 (14%) dead born.

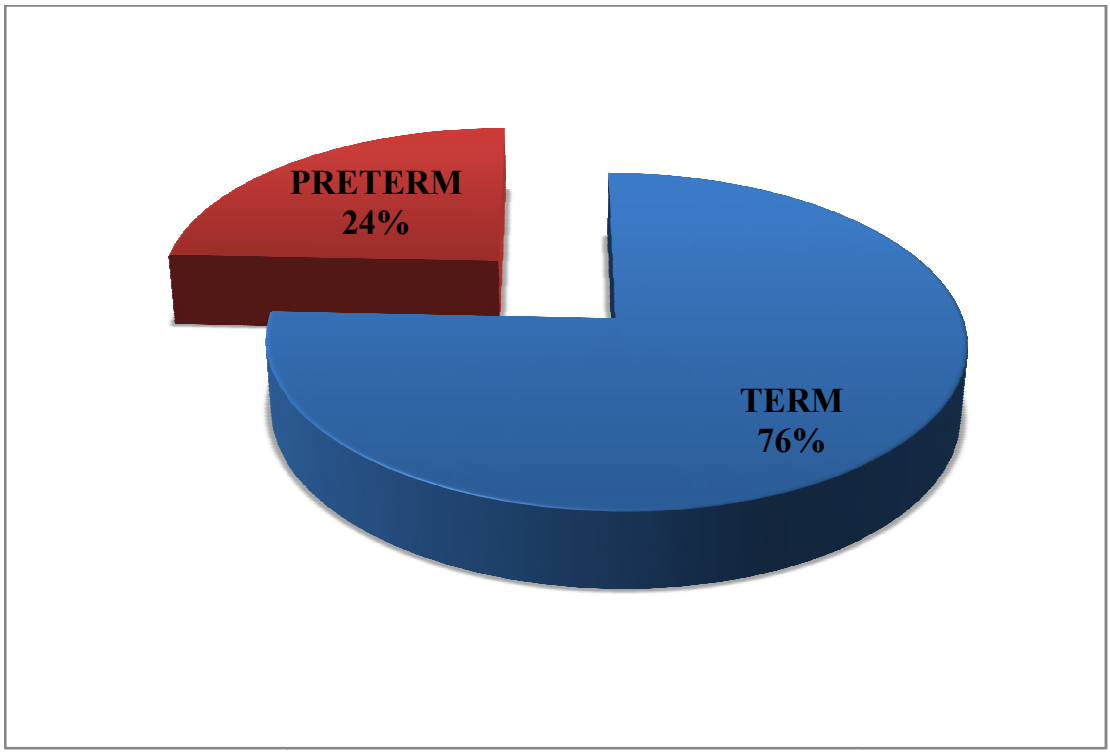
TABLE 13

MATURITY OF THE BABY

S.NO	MATURITY	TOTAL	PERCENTAGE
1	TERM	28	75.7
2	PRETERM	9	24.3
	TOTAL	**37**	**100**

CHART 13

MATURITY OF THE BABY



Of the 37 live births , 28(75.7%) were term and 9(24.3%) were preterm.

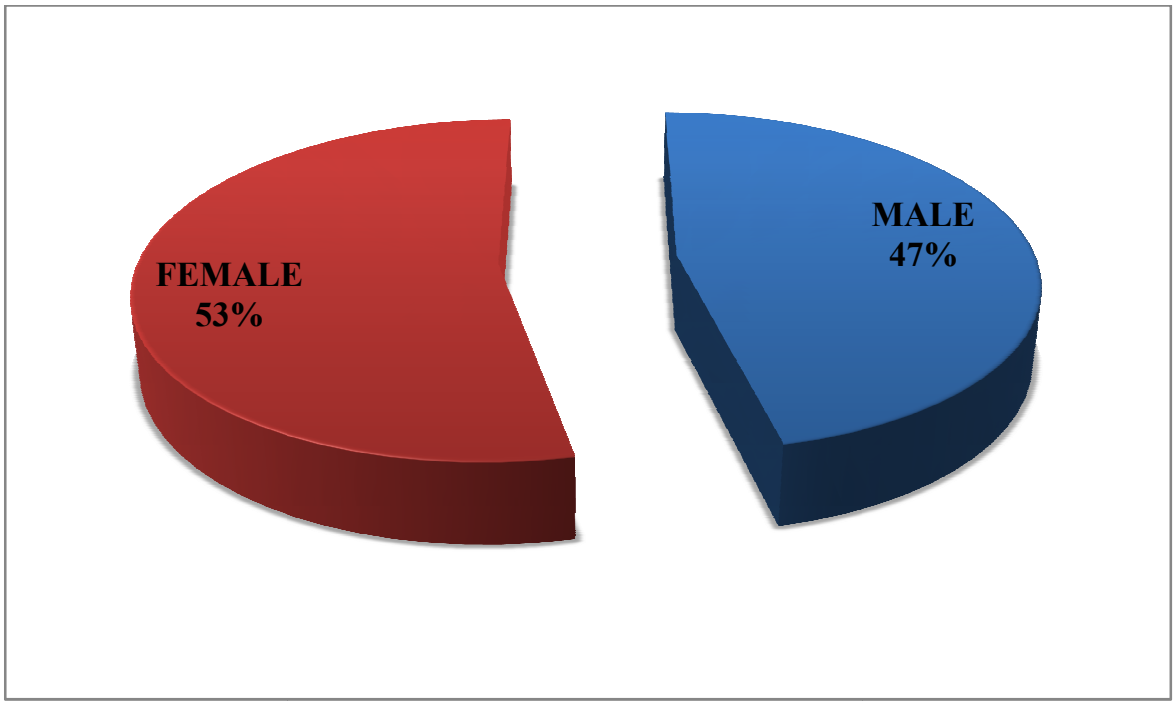
TABLE 14

SEX OF THE BABY

S.NO	SEX	TOTAL	PERCENTAGE
1	MALE	20	46.5
2	FEMALE	23	53.5
	TOTAL	**43**	**100**

CHART 14

SEX OF THE BABY



Of the 43 women delivered, 20(46.5%) were male,and 23(53.5%) were female.

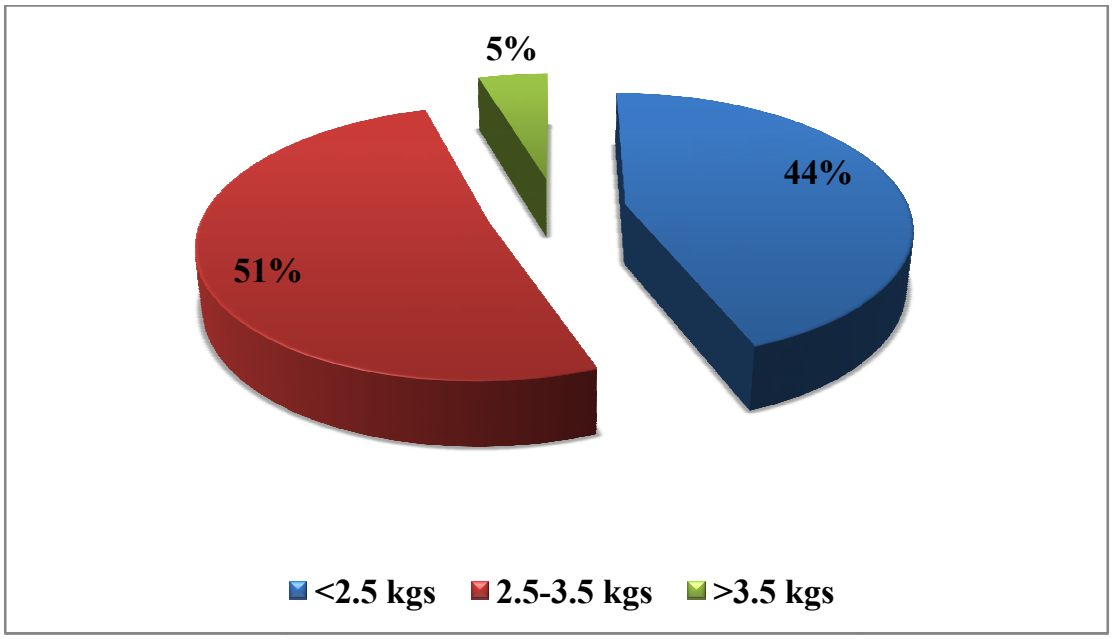
TABLE 15

BIRTH WEIGHT OF THE BABY

S.NO	BIRTH WT	TOTAL	PERCENTAGE
1	<2.5	19	44.2
2	2.5-3.5	22	51.2
3	>3.5	2	4.7
	TOTAL	43	100

CHART 15

BIRTH WEIGHT OF THE BABY



Low birth weight is common among jaundiced patients,44.2% had a birth weight <2.5 kg.only 4.7% had birth weight >3.5 kg.

TABLE 16

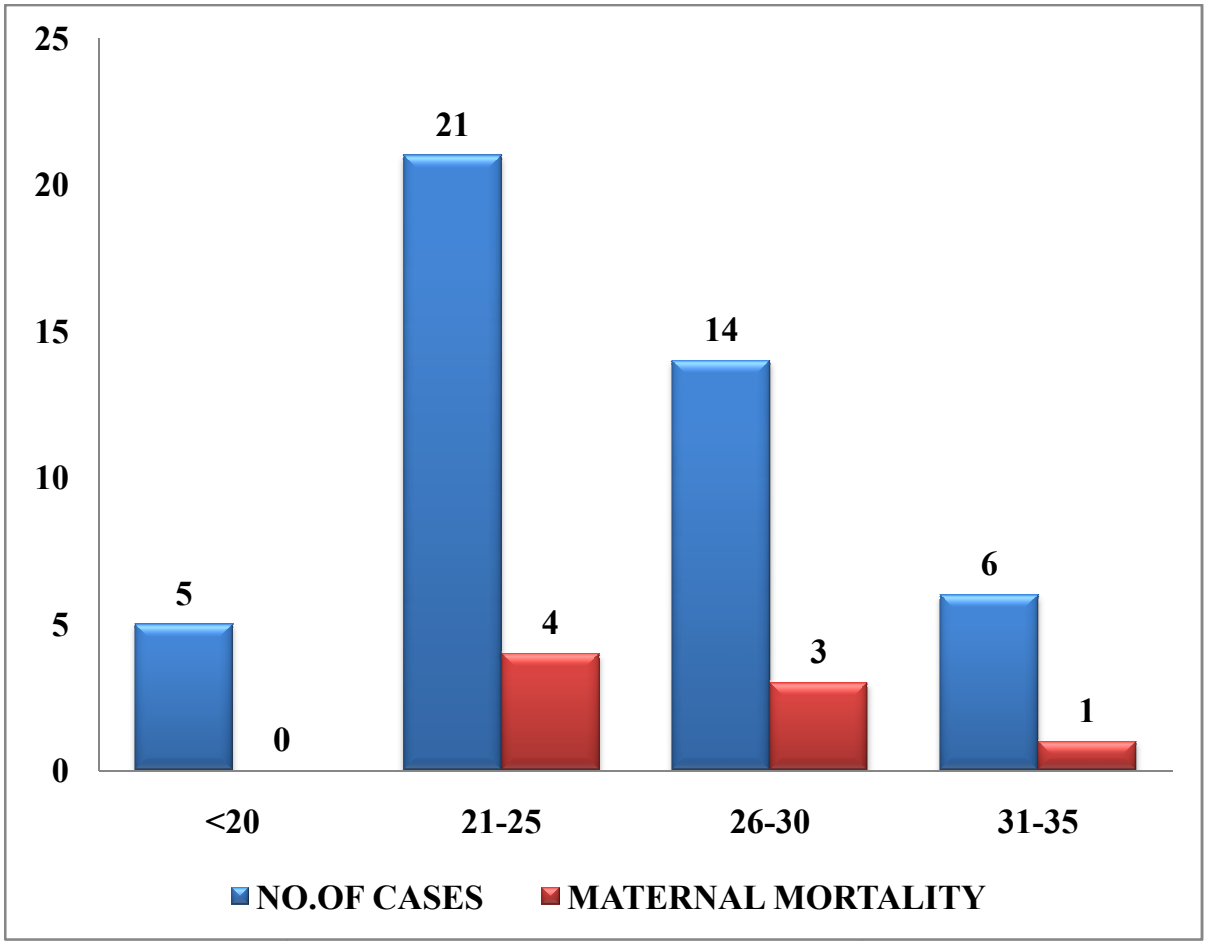
RELATION OF MATERNAL AGE TO

MATERNAL MORTALITY

S.No	Age group	No. of cases	Maternal mortality	% of mortality in relation to age
1	<20	5		0
2	21-25	21	4	50
3	26-30	14	3	37.5
4	31-35	6	1	12.5
	TOTAL	**46**	**8**	**100**

CHART 16

RELATION OF MATERNAL AGE TO MATERNAL MORTALITY



Maternal mortality is more common in the age group of 21-25 years, so do the number of cases.

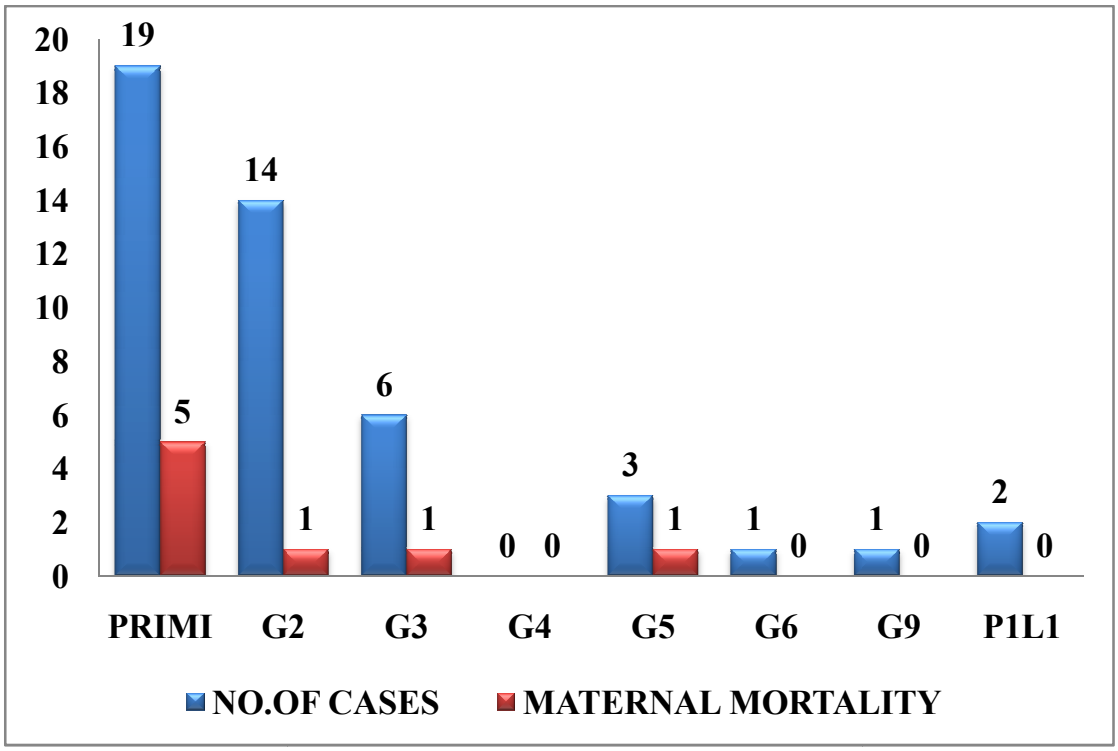
TABLE 17

GRAVIDITY IN RELATION TO MATERNAL MORTALITY

S.NO	GRAVIDITY	NO.OF CASES	MATERNAL MORTALITY	%OF MORTALITY
1	PRIMI	19	5	62.5
2	G2	14	1	12.5
3	G3	6	1	12.5
4	G4	-	-	-
5	G5	3	1	12.5
6	G6	1	-	-
7	G9	1	-	-
8	P1L1	2	-	-
	TOTAL	**46**	**8**	**100**

CHART 17

GRAVIDITY IN RELATION TO MATERNAL MORTALITY



Mortality is common in primigravida,out of 19 primi 5 died,giving an incidence of 62.5%.

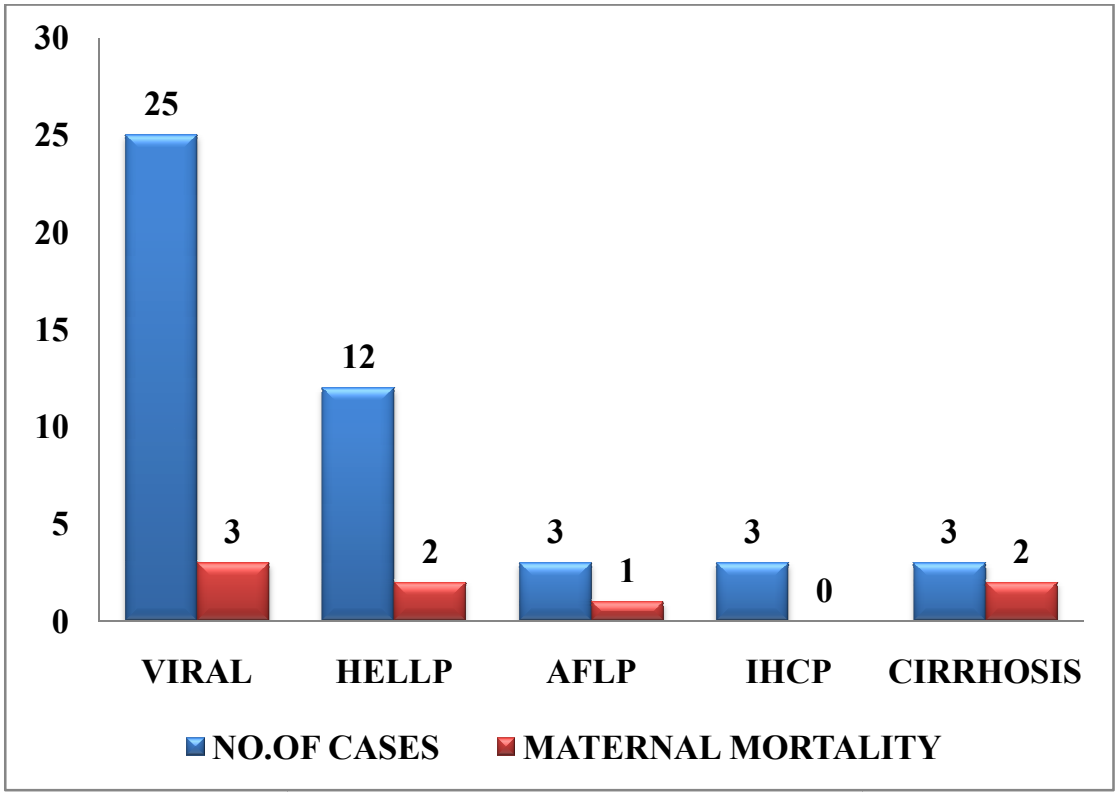
TABLE 18

ETIOLOGY IN RELATION TO MATERNAL MORTALITY

S.NO	ETIOLOGY	NO.OF CASES	MATERNAL MORTALITY	% OF MORTALITY
1	VIRAL	25	3	37.5
2	HELLP	12	2	25
3	AFLP	3	1	12.5
4	IHCP	3	-	-
5	CIRRHOSIS	3	2	25
	TOTAL	**46**	**8**	**100**

CHART 18

ETIOLOGY IN RELATION TO MATERNAL MORTALITY



Viral hepatitis is the most common cause of jaundice in pregnancy.25 out of 46 had viral hepatitis and 3 of them died(37.5%).HELLP and Cirrhosis contribute 25% in maternal mortality,12.5% contribution is from acute fatty liver of pregnancy.The maternal mortality and morbidity is low with intrahepatic cholestasis of pregnancy.

TABLE 19

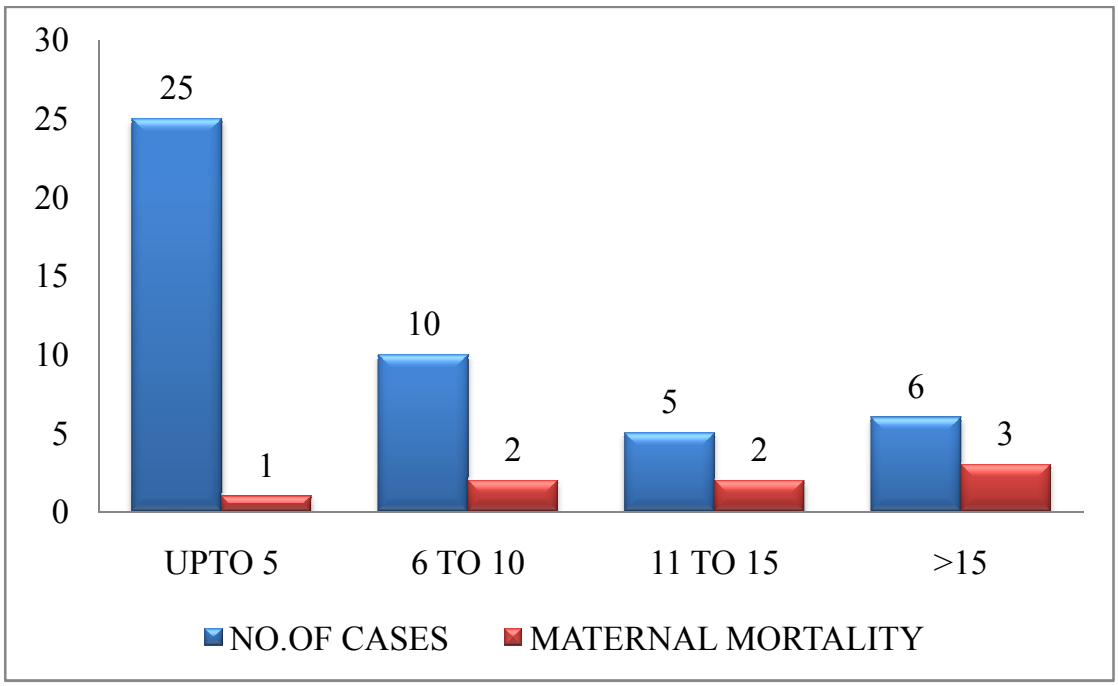
RELATION OF SERUM BILIRUBIN LEVELS TO

MATERNAL MORTALITY

S.NO	BILIRUBIN LEVELS	NO.OF CASES	MATERNAL MORTALITY	% OF MORTALITY
1	UPTO 5	25	1	12.5
2	6-10	10	2	25
3	11-15	5	2	25
4	>15	6	3	37.5
	TOTAL	**46**	**8**	**100**

CHART 19

RELATION OF SERUM BILIRUBIN LEVELS TO MATERNAL MORTALITY



6 patients had a serum bilirubin level of >15 and 50% of them expired.

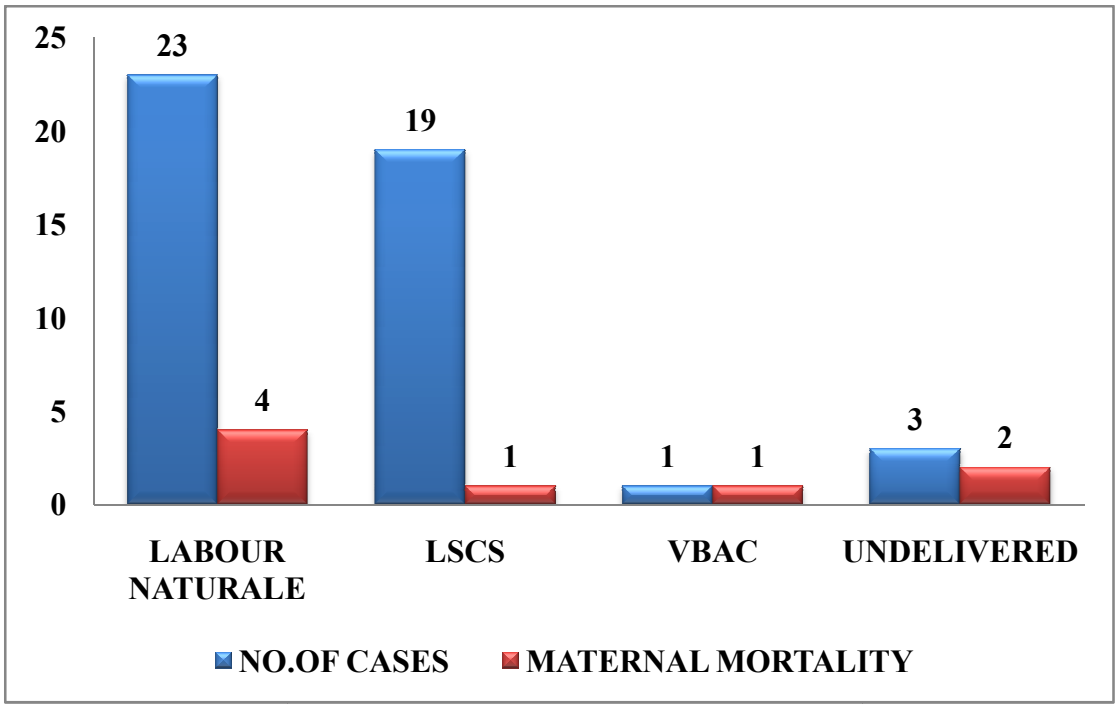
TABLE 20

RELATION OF MODE OF DELIVERY TO MATERNAL MORTALITY

S.NO	MODE OF DELIVERY	NO.OF CASES	MATERNAL MORTALITY	% OF MORTALITY
1	LABOUR NATURALE	23	4	50
2	LSCS	19	1	12.5
3	VBAC	1	1	12.5
4	UNDELIVERED	3	2	25
	TOTAL	**46**	**8**	**100**

CHART 20

RELATION OF MODE OF DELIVERY TO MATERNAL MORTALITY



Among 43 delivered, 23 had normal vaginal delivery,19 lscs and 1 VBAC.

DISCUSSION

The incidence of Jaundice among the pregnant women attending the Coimbatore Medical College Hospital, Coimbatore from August2013 to August 2014 is 3 per 1000 population. The incidence in various places by various authors is shown in the following table.

S.No.	Author	Year	Incidence per 1000 population.
1.	Sarkar et al (Calcutta)	1992	2.3
2.	Reddi Rani et al (Pondicherry)	1993	1.17
3.	Devinder Kaur et al (Delhi)	2011	0.92

Our hospital incidence correlates with the study of **sarkar et al**, 1992. Our hospital incidence is very high when compared to Western countries where it is 1 in 1500 pregnancies **(Williams 2010)**. This is because of higher prevalence of malnutrition, poor sanitation and low socioeconomic status in our country.

ETIOLOGY

In the present study viral hepatitis was found to be the commonest cause (54.3%) next being HELLP syndrome (26.1%) third in order is acute fatty liver of pregnancy, intrahepatic cholestasis of pregnancy, chronic liver disease such as cirrhosis with portal hypertension each contributing 6.5%. Patients with viral hepatitis was the major group with Jaundice in pregnancy constituting to 54.3%. 21 patients had acute hepatitis B infection and 4 were presumptively taken as type E . Jaiswal SP and Naik G in 2001 and Jain A, Sharma JK in 1999 analysed various types of viral hepatitis during pregnancy. Their studies were shown below:

Place	Author	Year	HAV	HBV	HCV	HDV	HEV	Noviral markers
Indore	Jaiswal SP Naik G Soni N	2001	7%	19%	4%	Nil	46%	24%
New Delhi	Jain A Sharma JK	1999	4%	28%	2%	Nil	42%	18%

Because of universal exposure during infancy and childhood, HAV infection is more common in children rather than in adults in developing countries like India. HDV always coinfects with Hepatitis B virus and cannot survive independently. Hence acute viral hepatitis in an adult in a

high prevalence country such as India is more likely to be HEV or HBV rather than HAV, HCV or HDV **(Michael de Swiet, 2002).**

AGE :

In the present study 45.7% of pregnant jaundiced women were between 20-24 years. This correlates with the study of **ShethAbhay et al, 1990, Devinder Kaur et al, 2001 and Kamala Jeyaram et al, 1988**. This age group is having the maximum fertility rate and maximum number of deliveries. Early age of marriage in lower socioeconomic group due to illiteracy also contributes. (In Western Countries, only 30% are under 24 years).

GRAVIDITY

In this study 41.3% of patients were primigravida; It correlates with the study of **Reddi Rani et al, 1993** and **J.S. Chauhan et al, 1983** (50%).**Kamala Jeyaram et al, 1986** and **Devinder Kaur et al, 2001** had showed only 30% incidence among primigravida.

GESTATIONAL AGE

93.5% of patients presented in 3rd trimester. This correlates well with the study of **C.M. Alwani et al., 1986**, **Reddi Rani et al, 1993**.

Increaed incidence in third trimester is probably due to excess nutritional stress in late pregnancy.

SOCIOECONOMIC STATUS

95% patients belonged to lower socioeconomic group in this study. This shows the influence of malnutrition, poor sanitation, water contamination, sexual abuse and poor health awareness in the development of jaundice as stressed by various authors.

BIOCHEMICAL PARAMETERS

In this study, 50% of maternal mortality were with serum bilirubin more than 15mg/dl. In the study of **Chadda et al, 1983** majority of mortality occurred with serum billirubin level more than 15mg. 50% of maternal mortality were observed with serum transaminase levels between100-400IU/L in our study. Mortality also reported with low level and this is due to exhaustion by previous excessive release of transaminase (Issel Backer 1987).

The prothrombin time is the most sensitive indicator of severity of liver dysfunction and hence the prognosis (**Weinstein et al, 1982**). A mildly elevated prothombin time usually indicates concurrent DIC whereas grossly elevated prothombin time signify significant hepatic

necrosis. **Khuroo M.S. & Kamili** in 2003 reported poor prognosis in patients with prothombin time > 30 secs.

COMPLICATIONS

12 patients had anaemia. Among them 2 had severe anaemia (Hb <7gm%) and rest of them were between 7-10gm%. Among the 2 one died of variceal bleed. Among 6 pre-eclampsia patients 3 died. Anaemia and preeclampsia in a jaundiced patient further worsens the prognosis.2 patients had PPH. Among 46 patients, 21 required blood transfusion. **Alwani et al, 1985** reported 31% incidence of post portum haemorrhage in his studies. Our study has significantly lower incidence of PPH (25%).

MODE OF DELIVERY

41.3% of patients were delivered by LSCS for obstetric indications and 52.2% had labour natural. Since most were preterm, labour was easier. Mode of delivery did not influence maternal or fetal outcome. Early delivery by caesarian section to improve maternal and fetal survival and arresting the disease is recommended by **Peter's et al, 1967** and **Burrough's et al., 1983**. However the risk of anaesthesia and bleeding due to coagulation abnormalities are the factors against it.

FETAL OUTCOME

Among the total 43 patients, 24.3% had preterm delivery and 75.7% had term deliveries. **Sheth Abhay et al, 1999** reported 3% incidences of abortion and 55% preterm deliveries whereas **Subodh Singh et al, 1991** reported 8% abortion and 64% preterm delivery.

Total number of fetal death is 12. Among these 11 were preterm babies and only 2 was term. All neonatal deaths were because of prematurity and low birth weight. Prematurity and low birth weight as a cause of neonatal mortality was stressed by various authors (**Jai Bagwan Singh, 1990, Subodh Singh et al, 1991, Devinder Kaur et al, 2001**). Fetal loss by abortion and prematurity may be due to high fever and general debility associated with high viraemia. Perinatal mortality is 24%.

MATERNAL OUTOME

Out of 46 jaundiced pregnant women in this study, 8 women died. 2 patients died of HELLP syndrome, 3 patients due to viral hepatitis, 2 due to cirrhosis with portal hypertension and 1 due to acute fatty liver of pregnancy.

HELLP Syndrome

HELLP Syndrome patients with jaundice constituted 12 patients, all of whom presented in late second and early 3rd trimester with severe hypertension. **Weinstein et al, 1985** has reported the mean gestational age of presentation of HELLP syndrome was 33.6 wks.Among 12 patients,8 normal term delivery, 3 were preterm delivery and one was intrauterine death. Perinatal mortality in this group was 50% (6 out of 12). **Weinsteinet al, 1985, Sibai et al, 1990** has reported a perinatal mortality rate of 60% in patients with HELLP syndrome. Higher perinatal mortality is due to prematurity. There was 2 maternal deaths in this group. One patient died due to DIC and other due to atonic pph . **Baha et al, 1990**, reported a maternal mortality of 24% in his study, our study correlates with it (25%)

Viral Hepatitis :

Out of 25 patients due to viral hepatitis, there were 3 maternal deaths. One patient had acute hepatitis B infection and she presented with term IUD. Her pregnancy was terminated by Labour naturale. Her prothrombin time and bleeding time was prolonged. Another patient had combined hepatitis e infection and went in for fulminant hepatic failure and DIC.

Maternal mortality in this group was 37%. Maternal mortality among pregnant women with viral hepatitis was 14.3% whereas is non pregnant women it was 5.6% **(De Swiet 2002)**. Age and parity did not reveal any significance.

In the studies reported by **Alwani et al, 1985** the cause of death among patients with viral hepatitis was hepatic coma in 2.4%, GI bleed in 15%, Hepatorenal syndrome in 15%, PPH in 13% and Hepatic encephalopathy in 8%. **Mirghani et al, 1992**, observed more than 80% of deaths occurred in postpartum period. It is concluded that pregnancy is a risk factor which increases the mortality of viral hepatitis.

Author	**Year**	**Abortion**	**Preterm delivery**	**Perinatal Mortality**
Medha et al	1993	8.3%	20%	27%
Padmaker	1986	8.3%	24%	30.9%
Alwani et al	1985	11.9%	18%	29%

IHCP :

There were 3 patients with intrahepatic cholestasis of pregnancy in the study. All presented with pruritis and Jaundice for 2-3 wks duration.There was no clinical evidence of viral hepatitis in these patients. One patient had term delivery and another one was pre term,the other intrauterine death. (**Rioseco AJ, 1994)** has reported an increased incidence of preterm labor (44% and 50% respectively) in IHCP patients.

Cirrhosis and Portal Hypertension :

In 3 patients with cirrhosis and portal hypertension, 2 died, in one of them splenic rupture occurred and taken up for laparatomy,, baby IUD and mother died 2 hrs after splenectomy, the other patient was a known portal hypertension patient with oesophageal varices, banding done 4 times,she was 11 wks pregnant when she was admitted with severe anemia and uncontrollable variceal bleeding. Inspite of multiple transfusions, she died.

MANAGEMENT

In patients with HELLP syndrome immediate termination of pregnancy once diagnosed is the treatment of choice, after stabilization of vitals. Strict control and frequent monitoring of BP is mandatory. Steroids also included in treatment.In IHCP, oral antihistaminic may provide some relief from pruritis, but UDCA is the preferred treatment. If patient is anicteric, pregnancy can be allowed to continue till term,otherwise pregnancy has to be terminated after completion of 36 weeks.

Management of patients with viral hepatitis was entirely symptomatic and supportive. It includes bed rest, high carbohydrate diet,glucose, vitamin B complex and IV dextrose. In none cases, termination of pregnancy was carried out prophylactically. Patients with chronic liver disease did not require any specific therapy except for frequent monitoring of liver function tests and coagulation profile. Blood and fresh frozen plasma were given when appropriate.

FETAL ANOMALIES

There was no congenital anomaly in the present study. **Shah** had reported that there is no convincing evidence that jaundice in the I trimester can cause fetal anomalies. So jaundice is not an indication for medical termination of pregnancies.

DEVELOPMENT OF NEONATAL JAUNDICE

4 babies had only physiological jaundice. A jaundiced mother giving birth to a jaundiced baby is rare because in most cases, conjugated bilirubin is present to which placenta is impermeable (**Sechar**).

SUMMARY

A study was made on 46 jaundiced mothers who attended obstetrics and gynaecology department to find out the maternal morbidity and mortality rates for a period of 1 year. The results came as

- Jaundice is more common in primigravida (41.3%).
- Common in the age group of 21-25 years (45.7%).
- The complications and incidence of jaundice is more common in third trimester (93.5%).
- Most common etiology for jaundice is viral hepatitis (54.3%),second common is HELLP syndrome.
- Death occurs most commonly due to DIC(37.5%).
- 21 patients had multiple blood transfusions.
- Out of 46 , 43 delivered and 3 undelivered, of the 43 cases 37(86%) were live born and 6(14%) intrauterine death.
- Of these 37 cases , 28 were term (75.7), 9 preterm (24.3%)., 20 male babies (46.5%), 23 female babies (53.5%).

CONCLUSION

1. According to my study, the incidence of jaundice complicating pregnancy in Coimbatore medical college is 3/1000.
2. Jaundice in pregnancy is associated with high maternal and perinatal mortality
3. Viral hepatitis is the commonest cause with about 25 cases.
4. Among these 25 cases 21 had hepatitis B and the rest 4 had hepatitis E infection.
5. The factors responsible for high maternal mortality in our country are malnutrition, poor personal hygiene, ignorance, delay in seeking medical advice,
6. High serum bilirubin level is associated with high mortality levels.
7. The incidence is high in primigravida and in third trimester
8. Perinatal mortality is high in jaundice complicating pregnancy, the cause being prematurity and low birth weight.

BIBLIOGRAPHY

1. Kuscu NK,Koyuncu F. Hyperemesis gravidarum:current concepts and management.Postgrad Med J 2002;78:76-79.

2. Fell DB,Dodds L,Joseph KS,Allen VM,Butler B.Risk factors for hyperemesis gravidarum requiring hospital admissions during pregnancy.Obstet Gnecol 2006;107:277-284.

3. Dann AT,Kenyon AP,Seed PT,Poston L,Shennan AH,Tribe RM.Glutathione S transferase and liver function in intrahepatic cholestasis of pregnancy and pruritis gravidarum.HEPATOLOGY 2004;40:1406-1414.

4. Dixon PH,Weerasekara N,Linton KJ,Donalson O,Chambers J,Egginton E,et al.Heterozygous MDR3 missense mutation associated with intrahepatic cholestasis of pregnancy:evidence for a defect in protein trafficking.Hum Mol Genet 2000:9:1209-1217.

5. Wasmuth HE,Glantz A,Keppeler H et al.Inrtrahepatic cholestasis of pregnancy:the severe form is associated with common variants of the Hepatobiliary phospholipid transport gene ABCB4.Gut 2007;56:256-270.

6. Glantz A,Marschall H-U,Mattsson L-A.Intrahepatic cholestasis of pregnancy;relationships between bile acid levels and fetal complications rates.HEPATOLOGY 2004;40:467-474.

7. Kondrackiene J,Beuers U,Kupcinskas L.Efficacy and safety of ursodeoxycholic acid versus cholestyramine in intrahepatic cholestasis of pregnancy.Gastroenterology 2005;129:894-901.

8. Binder T,Salaj P,Zinna T,Vitek L.Randomised prospective comparative study of ursodeoxycholic acid and S-adenosyl L methionine in the treatment of intrahepatic cholestasis of pregnancy.J Perinat Med 2006;34:383-391.

9. Pockros PJ,Peters RL,Reynolds TB.Idiopathic fatty liver of pregnancy:findings in ten cases.Medicine 1984;63:1-11.

10. Burroughs AK,Seong NH,Dojcinov DM et al.Idiopathic acute fatty liver of pregnancy in 12 patients.Q J Med 1982;51:481-497.

11. Rolfes DB,Ishak KG.Acute fatty liver of pregnancy:a clinicopathologic study of 35 cases.Hepatology 1985;5:1149-1158.

12. Borwning MF,Levy HL,Wilkins-Haug L,Larson C,Shih VE.Fetal oxidation defects and maternal liver disease in pregnancy.Obstet Gynecol 2006;107:115-120.

13. Yang Z,Zhao Y,Bennet MJ et al.Fetal genotypes and pregnancy outcomes in 35 families with mitochondrial trifunctional protein mutations.Am.J Obstet.Gynecol.2002;187:715-720.

14. Quigley MM.Acute obstetric yellow atrophy presenting as idiopathic hyperuricemia.South Med J 1974;67:142-144.

15. Goodin B.An overview of expanded newborn screening for inborn errors of metabolism.Practical gastroenterol 2006;41:34-51.

16. Bellig LL,Maternal acute fatty liver of pregnancy and associated risk for long chain 3-hydroxyacyl-coenzyme A dehydrogenase(LCHAD) deficiency in infants.Adv Neonatal care 2004;41:34-51.

17. Sibai B,Dekker G,Kupferminc M,Preeclampsia.Lancet 2005;365:785-799.

18. Rolfes DB,Ishak KG.Liver disease in toxemia in pregnancy.Am. J Gastroenterol 1986;81:1138-1144.

19. Manas KJ,Welsh JD,Rankin RA et al.Hepatic hemorrhage without rupture in preeclampsia.N.Eng.J Med .1985;312:424-426.

20. Bis KA,Waxman B.rupture of liver associated with pregnancy:a review of the literature and report of two cases.obstet.Gynecol.Surv.1976;31:763-773.

21. Rowan JA,North RA.Abnormal liver function tests after preeclampsia.Lancet 1995;345:1367.

22. Baxter JK,Weinstein L.HELLP syndrome:state of art.Obstet Gynecol surg 2004;59:838-845.

23. Barton JR,Sibai BM.Diagnosis and management of hemolysis,elevated liver enzymes and low platelet syndrome.Clin prenatal 2004;31:807-833.

24. Muetze S,Leeners B,Ortlepp JR et al.Maternal factor v leiden mutation associated with HELLP syndrome in Caucasian women.Acta Obstet.Gynaecol.Scand 2008;87:635-645.

25. Strand S,Strand D,Seufert R et al.Placenta derived cd95 ligand causes liver damage in HELLP syndrome.Gastroenterology 2004;126:849-858.

26. Sibai BM.Diagnosis,controversies and management of HELLP syndrome.Obstet Gynecol 2004;103:981-991.

27. Lu EJ,Curet MJ,El Sayed YY,Kirkwood KS.medical versus surgical management of biliary tract disease in pregnancy.Am Med J Surg2004;188:755-759.

28. Rollins MD,Chan KJ,Price RR,Laproscopy for appendicitis and cholelithiasis during pregnancy.susrg endos 2004;18:237-241.

29. Candia L,Marquez J,Epsinosa LR.Autoimmune hepatitis and pregnancy:a rheumatologists dilemma.Semin arthritis Rheum 2005;35:49-56.

30. Schsienberg IH,Jaffe ME,Sternlieb I.the use of trientine in preventing the effects of interrupting pencillamine therapy in Wilson's disease.N Engl J Med 1987;317:209-213.

MATERNAL OUTCOME IN JAUNDICE

COMPLICATING PREGNANCY

NAME: AGE: IPNO:

D.O.A:

D.O.DELIVERY:

D.O.DISCHARGE:

PARITY:

L.M.P: E.D.D:

COMPLAINTS:

HOPI:

PAST H/O:

MENSTRUAL H/O:

MARITAL H/O:

OBSTETRIC H/O:

ON EXAMINATION:

CONSCIOUS/ORIENTED - ANAEMIC

TEMPERATURE- PEDAL EDEMA-

ICTERIC – MILD/SEVERE

PR- BP- CVS-
RS-

P/A-

P/V-

INVESTIGATIONS:

HB% -

URINE ALBUMIN

SUGAR

DEPOSIT

BS/BP

BLOOD SUGAR

UREA

S.CREATININE

LFT

T.BILIRUBIN

DIRECT

INDIRECT

SGOT

SGPT

ALP

LDH

PLATLET COUNT

CLOTTING TIME

BLEEDING TIME

PROTHROMBIN TIME

ACTIVATED PARTIAL THROMBOPLASTIN TIME

VIRAL MARKERS:

ULTRASOUND:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் மகப்பேறு மருத்துவ துறையில் பட்ட பயிலும் மாணவி அவர்கள் மேற்கொள்ளும் **"மஞ்சள் காமாலை நோயால் தாய்க்கு ஏற்படும் உடல்நலக்குறைவு மற்றும் உயிர்சேதம்**" குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்தில் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

KEY TO MASTER CHART

U/A	–	Urine albumin.
U BS/BP	–	Urine bile salt/bile pigment
BT	–	Bleeding time
CT	–	Clotting time
PT	–	Prothrombin time
HB	–	haemoglobin .
SGOT	–	Serum glutamic oxaloacetic transaminase
SGPT	–	Serum glutamic pyruvic transaminase
SAP	–	Serum alkaline phosphatase.
HBsAG	–	Hepatitis B surface antigen
Anti HCV	–	Anti hepatitis c virus
HEV	–	Hepatitis E virus.

S: No	Name	Age	Parity	Gestation Age in Weeks	Etiology	Duration of Symptoms	U/A	UB/BP	BT	CT	PT	HB	Platelet	S.Bilirubin	SGPT	SGOT	SAP	HBSAg	ANTI HCV	HEV	Maternal Outcome				Fetal Outcome			
																					Transfusion	Mode of Delivery	Complications	Death / Recovery	Term / Preterm	Birth Wt in Kg	APGAR	Complications
1	Jerina Begam	28	primi	30	HELLP	2 wks	2+	+	3	8	28	9.8	3.8 lakh	7.3	98	131	171	-	-	-	nil	undelivered	encephalopthy	Death				
2	Maragatham	23	primi	39	Viral(HBV)	1 wk	nil	+	2	4	13	9.6	1.8 lakh	1.4	56	43	79	+	-	-	nil	LSCS	NIL	Recovered	Term FCH	3	8/10	NIL
3	Priya	24	primi	40	Viral(HBV)	3 days	nil	nil	2	3	12	11	2.1 lakh	2.2	75	73	266	+	-	-	nil	LN	NIL	Recovered	Term FCH	3.1	9/10	NIL
4	Prasanna	26	P1L1		cirrhosis with portal hypertension	3 days	nil	nil	3	4	12	8.2	82,000	1	22	26	441	-	-	-	1pc,4 ffp	LSCS	NIL	Recovered				
5	Guna Sundari	21	primi	36	AFLP	1 day	1+	2+	2	10	26	14	52,000	14.2	92	152	412	-	-	-	4 pc,8 ffp	LN	Atonic PPH	death	Term MCH	2.7	6/10	Died
6	Thilagavathy	21	G2P1L0	term	viral(HEV)	1 day	nil	+	3	3	12	11	2.4 lakh	1.3	78	84	112	-	-	+	nil	LN	NIL	Recovered	Term FCH	2.4	8/10	NIL
7	Dhanalakshmi	32	G2P1L1	28	Viral(HBV)	2 days	nil	2+	4	4	20	10.9	84000	0.8	86	92	141	+	-	-	1pc,4 ffp	LSCS	NIL	Recovered	IUD MCH			
8	Shiji	20	G2A1	34	IHCP	2 days	1+	1+	3	6	20	14.5	1.79 lakh	17.1	179	440	193	-	-	-	2pc	LN	Vulval hematoma	Recovered	Alive FCH pre term	1.8	6/10	NIL
9	Sangeetha	24	G3P1L1 A1	term	Viral(HBV)	1 day	nil	nil	2	5	26	10.6	2.11 lakh	3.8	142	112	186	+	-	-	nil	LN	NIL	Recovered	Term Alive MCH	2.7	8/10	NIL
10	Shanthini	27	G2P1L1	32	HELLP	9 days	1+	+	1	8	20	11	60,000	11.6	43	52	257	-	-	-	2pc,4ffp	LSCS	NIL	Recovered	Pre term FCH	1.8	6/10	NIL
11	Priyanka	20	primi	term	AFLP	2 days	nil	2+	3	7	13	8.7	53,000	12.3	109	79	385	-	-	-	1pc,4 ffp	LN	NIL	Recovered	IUD MCH	2.7		
12	Gandhimathi	32	G2P1L1	term	Viral(HBV)	8 days	nil	nil	2	4	12	10.5	1.7 lakh	1.1	26	28	140	+	-	-	nil	LSCS	NIL	Recovered	Term MCH	3.6	8/10	NIL
13	Ruth Priyadharshini	24	P1L1	day of delivery	HELLP	1 day	nil	nil	3	8	13	7.9	96,000	8.7	150	366	230	-	-	-	2pc	LN	NIL	Recovered	Term FCH	2.4	7/10	NIL
14	Shanthi	29	G5P3L1 A1	term	HELLP	2 days	1+	nil	4	4	11	11	70,000	7.8	260	272	1176	-	-	-	2pc	LSCS	NIL	Recovered	Term FCH	2.8	6/10	Died
15	Banumathy	31	primi	term	viral(HBV)	1 day	nil	2+	3	3	12	12	2 lakh	10.8	116	181	227	+	-	-	nil	LN	NIL	Recovered	Term FCH	3.1	8/10	NIL
16	Baby Kaneeshwari	26	primi	term	Viral(HBV)	2 days	nil	1+	2	4	13	10	1.11 lakh	6.4	21	18	123	+	-	-	nil	LSCS	NIL	Recovered	Term FCH	3.2	8/10	NIL
17	Sundarammal	27	primi	term	viral(HBV)	1 day	nil	1+	2	7	13	11	3.45 lakh	8.3	39	45	535	+	-	-	nil	LSCS	NIL	Recovered	Term MCH	2	7/10	Died
18	Chinna Marathal	33	G3P2L2	36	HELLP	1 week	2+	nil	3	8	13	5.1	7000	2.2	96	84	210	-	-	-	4pc,10plt, 12ffp	VBAC	DIC	Death	preterm mch	1.8	7/10	Died
19	Anbuselvi	22	G5P1L1 A3	37	Viral(HBV)	1 day	nil	nil	3	7	12	10.4	1.9 lakh	1.6	31	27	130	+	-	-	nil	LN	NIL	Recovered	Alive MCH term	2.6	8/10	NIL
20	Bhuvaneswari	26	primi	37	cirrhosis with portal hypertension	1wk	nil	nil	4	7	13	10.1	3.4 lakh	0.9	98	72	112	-	-	-	4pc,10ffp	LSCS	splenic rupture	Death	IUD MCH	1.9		
21	Devi	29	G3P2L1	29	IHCP	1 day	nil	nil	2	6	12	11.8	2.21 lakh	18.3	110	106	152	-	-	-	nil	LN	NIL	Recovered	IUD MCH	1.2		
22	Pushpa	21	G9P2L1 A6	38	IHCP	2 wks	nil	nil	3	7	13	10	2.3 lakh	4.6	49	55	311	-	-	-	nil	LSCS	NIL	Recovered	Term FCH	2.1	7/10	NIL
23	Madhuri Devi	23	G2P1L0	40	Viral(HBV)	-	nil	nil	2	4	12	13.5	69,000	3.4	24	26	86	+	-	-	nil	LSCS	NIL	Recovered	Term FCH	2.6	8/10	NIL
24	Kalishrani	22	primi	28	viral(HBV)	-	nil	nil	3	7	14	7.8	1.8 lakh	8.2	299	210	814	+	-	-	nil	LN	NIL	Recovered	Pre term MCH 1.8 kg	1.2	7/10	Died

S: No	Name	Age	Parity	Gestation Age in Weeks	Etiology	Duration of Symptoms	U/A	UB/BP	BT	CT	PT	HB	Platelet	S.Bilirubin	SGPT	SGOT	SAP	HBSAg	ANTI HCV	HEV	Maternal Outcome				Fetal Outcome			
																					Transfusion	Mode of Delivery	Complications	Death / Recovery	Term / Preterm	Birth Wt in Kg	APGAR	Complications
25	Chellammal	20	primi	term	HELLP	3 days	nil	nil	5	8	20	8	92,000	11.1	86	124	298	-	-	-	2pc,10ffp	LN	NIL	Recovered	Term FCH	2.8	8/10	Died
26	Malathy	26	G2A1	term	Viral(HBV)	4 days	nil	nil	3	7	18	10.4	2.87 lakh	2.6	29	27	135	+	-	-	nil	LSCS	NIL	Recovered	Term FCH	3.6	9/10	NIL
27	Kalamani	24	G2P1L1	term	HELLP	2 days	nil	nil	2	3	20	5.1	32,000	7.1	34	37	90	-	-	-	1pc, 2plt	LN	Atonic PPH	Death	Term FCH	2.4	7/10	NIL
28	Gomathi	25	G3P2L2	term	HELLP	4 days	nil	nil	3	6	24	7.6	90,000	6.7	128	114	370	-	-	-	2pc,6plt, 4ffp	LSCS	NIL	Recovered	Term MCH	2.6	8/10	NIL
29	Selvi	34	G2P1L1	term	HELLP	1 wk	2+	nil	2	5	20	8.1	76,000	5.8	270	240	860	-	-	-	2pc,2plt	LSCS	NIL	Recovered	Term MCH	2.5	7/10	NIL
30	Lavanya	26	primi	34	Viral(HBV)	2 wks	nil	+	3	5	18	10.1	1.86 lakh	4.4	178	264	1176	+	-	-	nil	LN	NIL	Recovered	Pre Term FCH	1.7	6/10	Died
31	Saranya	23	primi	term	viral(HEV)	1 wk	nil	2+	3	10	30	12.6	4.22 lakh	15.7	123	161	593	-	-	+	10ffp	LN	DIC	Death	Term IUD MCH	3		
32	Kanniyammal	27	primi	term	Viral(HBV)	2 days	nil	nil	3	6	20	11.8	2.18 lakh	2.4	56	58	114	+	-	-	nil	LN	NIL	Recovered	Term MCH	2.9	8/10	NIL
33	Rajeswari	25	G6P5L3	31	Viral(HBV)	1 wk	nil	nil	3	7	17	9.4	1.88 lakh	7	112	88	199	+	-	-	1pc,	LN	Atonic PPH	Recovered	Pre Term FCH	1.9	7/10	Recover ed
34	Prema	28	G2P1L1	32	Viral(HBV)	1 day	nil	1+	4	6	13	10.2	2.86 lakh	2.4	186	172	286	+	-	-	nil	LSCS	NIL	Recovered	Pre term MCH	1.9	8/10	Recover ed
35	Radha	31	G2P1L1	29	viral(HBV)	2 wks	nil	nil	2	4	13	12.8	1.44 lakh	2.4	26	28	91	+	-	-	nil	LN	NIL	Recovered	Pre Term Alive MCH	1.6	6/10	Recover ed
36	kalyani	26	primi	11	cirrhosis with portal hypertension	1 month	nil	+	3	16	30	2	1.86 lakh	18.1	178 6	186 6	2500	-	-	-	1pc,2ffp	undelivered	Variceal Bleed	Died				
37	Chithra	24	G3P2L2	34	viral(HBV)	1 day	nil	nil	1	5	12	10.6	2.81 lakh	7.6	88	92	126	+	-	-	nil	LN	Prerenal ARF	Recovered	Pre term FCH	1.8	7/10	NIL
38	Ramathal	20	primi	38	AFLP	3 days	1+	nil	4	6	20	11.2	79,000	4.8	72	86	119	-	-	-	nil	LSCS	DIC	Recovered	Term MCH	2.7	8/10	NIL
39	Bhooma	25	G2P1L1	39	Viral(HBV)	1 day	nil	nil	3	7	13	12.1	2.71 lakh	0.9	26	28	40	+	-	-	nil	LN	NIL	Recovered	Term FCH	2.5	7/10	NIL
40	Devi	23	primi	38	Viral(HBV)	3 days	nil	nil	2	6	12	10.6	1.98 lakh	2.4	28	27	41	+	-	-	nil	LSCS	NIL	Recovered	Term MCH	2.8	8/10	NIL
41	Lakshmi	24	G3A2	35	viral(HEV)	2 days	nil	nil	3	6	13	11.2	2.25 lakh	3.6	72	78	121	-	-	+	nil	Undelivered	NIL	Recovered				
42	Kala	26	G2P1L1	40	HELLP	1 day	1+	nil	3	5	18	10.8	66,000	2.1	86	88	562	-	-	-	2ffp, 2plt	LSCS	DIC	Recovered	Term FCH	3	8/10	NIL
43	Shylu Priya	25	G2A1	38	Viral(HBV)	2 days	nil	nil	2	6	12	9.4	1.72 lakh	2.6	72	76	112	+	-	-	nil	LN	NIL	Recovered	Term MCH	2.2	7/10	NIL
44	Sumathi	20	primi	37	HELLP	1 day	1+	nil	1	7	15	9.8	72,000	3.1	56	56	189	-	-	-	1pc,2plt, 4ffp	LN	NIL	Recovered	Term FCH	2.8	8/10	NIL
45	Dhanalakshmi	22	primi	39	HELLP	1 day	1+	nil	4	6	14	9.7	88,000	2.9	70	66	163	-	-	-	2plt,4 ffp	LSCS	NIL	Recovered	Term FCH	2.3	7/10	NIL
46	Latha	21	G5A4	34	viral(HEV)	1 day	nil	2+	3	5	28	11	1.11 lakh	13.6	88	186	440	-	-	+	8ffp	LN	DIC	Death	Term FCH IUD	2.9		